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THE REMOVAL OF AIR FROM THE RESPIRATORY TRACT AND CERTAIN OTHER BODY SPACES UNDER NORMAL AND ABNORMAL CONDITIONS

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The physiology and physics of the movement of oxygen and other gases in the lungs seem to be well understood. The partial tensions of the gases in alveolar air have been measured and the observed physiologic steps are consistent with the known laws of physics. Removal of entrapped air under pathologic conditions in the lungs and elsewhere is not so completely understood. For instance, it seems to be generally held that the air from a lung portion which becomes atelectatic is removed by absorption alone. It is assumed that a plug of mucus of such great viscosity that the cilia are unable to handle it corks a bronchus and that the air behind it is completely absorbed.[5, 9-11] Sometimes the opinion is ventured (seemingly without any direct evidence) that ciliary action is subnormal or has ceased entirely.

In many respects, this view appears to be essentially correct. There seems to be no doubt that air can be removed from an ob-

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structed lung by absorption alone. This happens in the presence of tumors and foreign bodies and has been demonstrated experimentally.[1-3] There are, however, discrepancies which make one suspect that other factors are involved. Pathologists and endoscopists sometimes find masses of very viscid mucus, but, more often, it is soft. The negative pressure associated with atelectasis may be considerable—34 mm of mercury has been measured. It is sufficiently strong to move the mediastinum to one side or the other and to practically immobilize the chest wall. One would suppose that the soft mucus would slide down into the area of negative pressure, but this it does not do. The absorption of air experimentally requires 16 hours[1-3] and postoperative atelectasis develops much more readily than that; surgeons have told me that it develops on the table. Thick, viscid casts of mucus are formed in asthma and are actually incorporated in the bronchial wall, but this results in emphysema much more frequently than in atelectasis. That cilia cannot handle viscid mucus appears to be in error also. Tests which I made in the open frontal sinus of an anesthetized dog indicated that viscid mucus can be removed more readily than very thin mucus. In these experiments, it was lifted en masse from the bottom of the sinus and carried to and through the ostium, occluding the latter completely during its passage.

It has been taught that the bronchial tree increases its cross-sectional area with each subsequent branching and that a composite bronchial tree would look like an inverted funnel. If this concept were accurate it might be possible that a viscid plug of mucus, carried upward in the bronchial tree, would become lodged in the narrowed stream bed. We made some measurements, however, of the bronchial tree which indicated that the concept of the inverted funnel applied to the bronchial tree is not correct, at least in bronchi larger than 1.5 mm.[7]

Theoretically, according to the known laws of physics, would it be possible for the air to be completely absorbed from an obstructed lobe subjected to a negative pressure of the magnitudes found in atelectasis?

It may be of value to review the steps by which air is absorbed from different portions of the body, in the light of the known laws of physics. We naturally think of the lungs first. The tidal respiratory flow while at rest is about 500 cc; 150 cc of each breath remains in the dead space of the bronchial tree; the other 350 cc flow into a residual volume of 2500 cc. There is some question that the air of any given quiet respiration actually reaches the alveoli proper; it is thought by some that it progresses only as far as the

alveolar ducts, atria or air sacs, whence it travels subsequently by diffusion into the alveoli. In any case, a rather complex situation is set up due to the differences in the partial gas tensions in alveolar air and atmospheric air. Diffusion is a necessary step in bringing the fresh oxygen to the alveolar wall where exchange of gases with the blood takes place. Oxygen molecules move at the rate of about 200 meters per second at room temperature; however, they bump into other molecules so frequently that the diffusion rate is very much smaller.

A physicist friend has given me the following formula for the diffusion of air at standard conditions of temperature and pressure: r=3.5 x the square root of t ("r" represents the average mean distance in centimeters traveled by the molecules as though they emerge from a point and move outward in all directions without obstruction and "t" is the time in seconds). Working this out, r=about 35 mm per second. The alveoli are approximately 0.2 mm in diameter or 0.1 mm in radius. This means that, during the one second which the air remains in the alveolus during inspiration, an oxygen molecule starting from the center of an alveolus could make the trip to the wall about 350 times. Even if it had to make the longer journey from the atria, there would still be ample time for a great many trips. The picture then of the gases from the inflowing air is that of the contained molecules flying rapidly in all directions and making contact with the walls of the alveolus from 1 to 400 times a second. The rate of absorption into the blood would depend upon, first, the nature of the interposed membranes, second, the comparative pressures of the gases in the blood and in the alveolus and, third, the relative solubility of the gases in the blood plasma.

Let us take a simpler example, for the moment, than that of the alveolus. In cataract surgery, we sometimes inject air into the anterior chamber at the end of operation in order to prevent adhesions between the iris and the incision. From four to six days are required for such a bubble in the anterior chamber to be absorbed. Let us follow the steps, as we see them, according to the known laws of physics (Fig. 1):

The partial gas pressures in the atmosphere are given about as follows: oxygen 158 mm of mercury, carbon dioxide 0.03 mm, nitrogen 597 mm and water vapor 5 mm (water vapor, of course, varies widely) making a total of 760 mm of pressure. The partial pressures for the aqueous are between those of the arterial and the venous blood; oxygen is given at 40-45 mm and carbon dioxide about 45 mm. The aqueous is produced in the ciliary body through a combination of dialysis, filtration and secretion. Oxygen, nitrogen

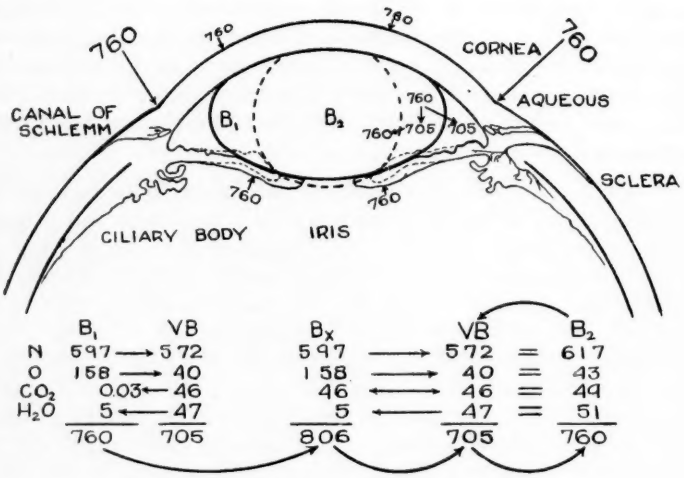

Fig. 1.—Diagram representing an air bubble injected into the anterior chamber of the eye. The figures in the column under B_1 are the partial pressures of the various gases contained in the injected air. Column VB gives the values in venous blood. It is assumed, for the purposes of this study, that the values are the same in the aqueous humor of the eye, although they probably differ somewhat. Since CO_2 diffuses more rapidly than the other gases, this gas would pass from the aqueous into the bubble faster than O_2 would dissolve. Therefore, at first, the pressure would increase, as indicated in Bx, if the volume should remain unchanged. The exchange of the other gases would soon take place as indicated between Bx and VB. Equilibrium would be established at 705 mm of pressure—if the volume should remain constant. The volume, however, does not remain constant since the yielding walls of the eye are subject to an atmospheric pressure of 760 mm. The volume would shrink until the pressure was equalized. Then the gas tensions would all be too high, as in B_2, and movement of molecules would continue into the aqueous, again causing a further fall in pressure. Equilibrium could not be attained and all of the air would be absorbed.

and carbon dioxide molecules are roughly about the same size as water and would, therefore, probably pass through the capillary walls, the stroma and the endothelium of the ciliary body about as rapidly as water, and, therefore, the concentrations would be much the same as in arterial blood. (Again, a reservation must be made, remembering that filtration may be interfered with by the processes of secretion.) The aqueous serves to nourish the lens, portions of the cornea and iris and probably other structures as well. In this metabolism, it undoubtedly loses oxygen and picks up carbon dioxide, as in the metabolism of tissues everywhere. It leaves the eye by several different routes, including the canal of Schlemm, through which the aqueous is in direct contact with venous blood with no intervening membranous barrier.

For our purposes, therefore, may we assume that the gas tensions present in the aqueous are the same as those in the venous blood. Those given for the venous blood are: oxygen 40 mm of mercury, carbon dioxide 46 mm, nitrogen 572 mm and water 47 mm, making a total of 705 mm, or 55 mm less than that of the atmosphere. The following steps would occur during absorption of the air bubble in the anterior chamber: The 0.03 mm of carbon dioxide would be rapidly increased to 46 mm, because the movement of carbon dioxide is some 35 times as fast as that of oxygen. Meanwhile, the 158 mm of oxygen would be reduced more slowly to 40 mm. The bubble would first increase in size or pressure because of the rapid movement of the carbon dioxide; the nitrogen moves much more slowly than either oxygen or carbon dioxide and, for purposes of simplification, can be assumed to stand practically still. The carbon dioxide, having reached equilibrium before the oxygen moved much, would soon be at a higher tension in the bubble as the oxygen moved out. If the bubble remained constant in size, the carbon dioxide would move back into the aqueous and the oxygen would move into the aqueous until equilibrium would be established with a net loss in pressure of 72 mm. However, since the eye is subjected to atmospheric pressure, disregarding for our purposes the intra-ocular pressure, the air bubble would not remain the same in size, but would shrink until the pressure again equalled 760 mm. As soon as this happened, then the tension of the carbon dioxide, oxygen and the nitrogen also would be greater in the bubble than in the aqueous, so all three would again move into the aqueous—the carbon dioxide very rapidly, oxygen much more slowly and the nitrogen still more slowly. This would again be followed by shrinkage of volume and the whole process would be repeated. Equilibrium would never be established and eventually all of the gases in the bubble would be dissolved in the aqueous.

Taking another example from our field of work, negative pressure sometimes develops within the sinuses. Assuming that the ostium should become completely blocked while the sinus was still full of air, through what physical steps would the gases of the air pass and what would be the eventual pressures? Conditions in the sinuses are far different from those in the alveolus of the lung or those in the anterior chamber of the eye. In the eye, the air is in direct contact with the aqueous, there being no interposing membranes. In the alveolar wall, there is at least one interposing membrane, namely the endothelium of the capillary between the alveolar air and the blood stream and probably, usually, a second one also, namely the endothelial lining of the alveolus. In the sinus, there is the capillary wall, a connective tissue stroma and a cuboidal type of epithelium, which is at least two cells deep. It would seem that these interposing structures would increase the time of the passage of gas molecules from the interior of the cavity into the blood stream. Moreover, the blood supply to the sinuses is normally very meager, and still another factor is that the sinus wall carries both oxygenated arterial blood and venous blood, while all of the blood entering the alveolar walls is venous blood.

Despite these handicaps, carbon dioxide would eventually find its way from the capillaries into the sinus cavity, oxygen would move slowly from the cavity into the venous blood, and, very much more slowly, nitrogen would also find its way into the blood stream. The total pressure within the sinus would rise at first because of the inflow of carbon dioxide molecules, but eventually it would begin to fall, as was the case in the eye. However, the sinus walls being rigid, there would be no decrease in volume and, therefore, the pressure, after the initial rise, would fall progressively until equilibrium would be established. If there were only venous blood present in the capillaries, the pressure theoretically would fall to 705 mm of mercury, the same as the gas pressure of the venous blood. However, the gas pressure of the arterial blood is 757 mm, therefore, the gas molecules would escape from the arterial blood into the cavity as they were removed from the cavity into the venous blood. Theoretically, equilibrium should be established at a pressure somewhere between 705 and 757 mm, probably in the neighborhood of 725 mm. The volume would remain the same as when closure of the ostium began (excluding, of course, such things as edema).

As a further illustration, the middle ear furnishes an example of conditions between those in the eye and in the sinus. Herbert[6] of Upsala, on introducing rubber balloons into the nasopharynx and inflating them in such a way as to close the eustachian tubes, has

found that in about 30 minutes there is a marked retraction of the eardrum, which he assigns to the reduced pressure following oxygen absorption. Here are conditions midway between those in the eye, where there is an external positive pressure and yielding, soft walls, and those of the sinus, where the walls are rigid and there can be no reduction in volume. That portion of the lateral wall of the middle ear which is comprised of the tympanic membrane is more or less yielding and the volume of the space is reduced somewhat. Therefore, the air in the middle ear would be more completely absorbed than that in the sinus. Still it is not possible for all of the air* to be absorbed; equilibrium would eventually be established at a pressure somewhere between the gas pressures of the arterial blood and the venous blood. The time required, as in the case of the sinus, would be long compared with that of the lung.

In the thorax, too, the tissues about an obstructed lobe of the lung are not free to yield as completely as those about the bubble in the eye (Fig. 2). The wall of the thorax is more or less rigid and a negative pressure develops within it. However, absorption can go on until the negative pressure equals the difference between the gas pressures in the alveolus and the gas pressures in the venous blood. This difference approximates 55 mm of mercury. Theoretically then, there could be established a negative pressure of 55 mm of mercury before equilibrium would be established and the absorption of gases would cease. The greatest negative pressure which has been measured in collapse of the lung is about 34 mm of mercury,[4] so, theoretically, all of the air from an obstructed lobe of the lung could be completely absorbed, provided 55 mm of negative pressure is not exceeded. However, a certain amount of time is required for absorption of gases, even from the lung. Coryllos and Birnbaum[2] report experiments in which individual gases were injected into the obstructed lung of a dog (i.e., a portion of the lung had been tied off). Absorption was accomplished for air in 16 hours, nitrogen 16 hours, oxygen 15 minutes, carbon dioxide 4 minutes, hydrogen 18 hours and helium 26 hours. As these experiments indicate, nitrogen and air require about the same amount of time for absorption.

Attempts were made to compare the time of absorption from the lung with that of the anterior chamber of the eye. The volume of the anterior chamber of the eye has been estimated to be about 1/20,000th of that of the lung. The volume of blood flowing through the lungs at rest is estimated at about 4600 cc per minute; through 1/20,000th part of the lung, this would be 0.23 cc or 230 cmm. The

*Again excluding such things as secretion, edema and other changes in the lining epithelium.

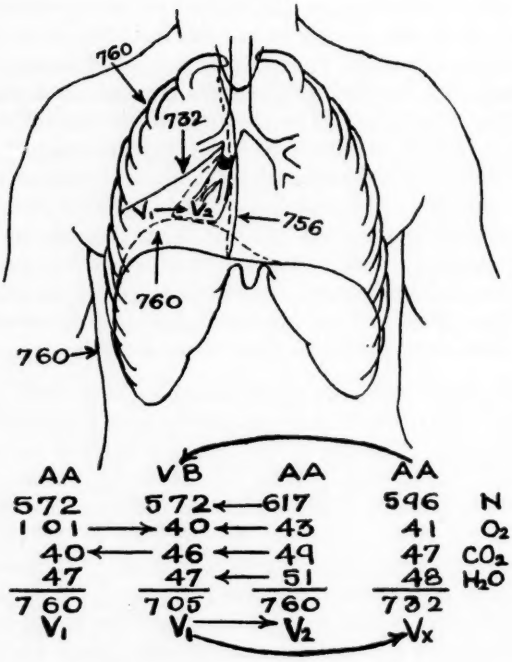

Fig. 2.—Absorption of air from an obstructed lobe of the lung and the effect of negative pressure. AA represents the gas tensions in alveolar air and VB the gas tensions in venous blood. When a lobe becomes obstructed exchange of O_2 and CO_2 would take place as indicated between AA and VB. If the volume (V_1) remained unchanged, equilibrium would be established at 705 mm. It would not remain constant, however. It would yield to the pressure of 760 mm (atmospheric pressure) in the surrounding tissues and shrink to V_2. All of the values would then be too high and gas molecules would pass from AA_1 to VB. If a negative pressure of 732 mm should develop within the thorax, as a result of shrinkage of the lobe (such pressures have been measured), then there would be further shrinkage in volume until the pressure within the lobe was also 732 (V_X). The gas tension would still be somewhat above venous blood and would move from AA_2 to VB. Equilibrium would not be possible unless the negative pressure within the thorax should drop to 705 mm (venous blood) or below. If the intrathoracic pressure should remain above 705 mm, theoretically absorption could be complete.

rate of circulation of aqueous through the eye is unknown; many attempts have been made to measure it, but the results vary from 2 to 40 cmm a minute. There are several other factors which make comparison difficult. Although there is no interposing membrane between the bubble and the aqueous in the eye, the bubble is all in one mass and exposes the minimum of surface for absorption, whereas an equal volume of air in the lungs is divided into hundreds of little spheres, each of which is surrounded by blood flow, presenting an area of about 500 sq. cm. In the aqueous, there are no blood cells to aid in the quick pick-up of the gas molecules, as there are in the blood surrounding the alveoli. It is difficult also to compare the time of absorption in the sinuses and ear with that in the lung; in the former, the air mass exhibits a small area for absorption compared with the latter. Moreover, the blood flow is only a small fraction of that in the lungs and the interposing membranes are very much denser and thicker. If 16 hours are required to absorb a volume of air from an obstructed lobe of a lung, probably several days would be required to absorb an equal volume from an obstructed sinus.

To summarize, air injected into the anterior chamber of the eye absorbs completely, but the time is comparatively long; it takes four to five days to absorb 0.1 cc. It requires several days for air to be completely absorbed from the pleural cavity or the subcutaneous tissue and several weeks from the peritoneal cavity. The only experiments bearing directly on the time factor in the case of the lungs, which I have been reported, are those by Coryllos and Birnbaum, where 16 hours were required to absorb air from the normal lung of a dog. These facts concerning the time of absorption would seem to indicate that some other factor besides absorption is acting in the removal of air in cases of postoperative atelectasis, which develop very promptly after operation.

In all of the articles in the literature on postoperative atelectasis, the matter of ciliary action is almost entirely overlooked. It seems to me that ciliary action cannot be ignored in the respiratory tract any more than heart action can be disregarded in the circulation or peristalsis in studying the physiology of the gastro-intestinal tract. There is no evidence that ciliary action in postoperative atelectasis is subnormal.

I performed experiments some years ago to determine the possible relationship of ciliary action to postoperative atelectasis. Nineteen experiments were done on the tracheas of freshly killed hens. Occluding masses of mucus were passed through these tracheas by ciliary action while the lower end was connected with a water manometer. Negative pressures developed almost immediately reaching a maximum in about

20 minutes; these pressures varied from 5 to 40 mm of water. In order to be certain that this phenomenon was not due to absorption of oxygen in the fresh tissue, another series of experiments was performed connecting the laryngeal or upper end of the trachea to the manometer. In each of these instances a positive pressure developed of about the same magnitude, namely up to 40 mm of water. Having in mind the probability that many masses of mucus in the longer tubes of a patient suffering from atelectasis might produce a cumulative effect, resulting in a higher pressure, another series of experiments was performed.[8] Three, and sometimes four, tracheas were connected in tandem and each was connected to a water manometer at its upper end. Connections which could be opened and closed at will were made so that the pressure resulting in each trachea could be individually measured independently of the others and, when desired, all of them could be connected as a single tube and the cumulative pressures recorded. These experiments demonstrated clearly that the effect is cumulative and pressures as high as 150 mm were obtained in this way.

Several series of experiments were done on the frontal sinuses of dogs. In the first of these, two needles were forced into the frontal sinus of an anesthetized dog; one was connected to a water manometer and through the other a quantity of mucus was injected. A negative pressure began to develop after a few minutes, reaching a maximum in about 20 minutes; some of these pressures were as high as 60 mm of water. In order to rule out the factor of absorption, a second series was done in which the dog was bled to death through the femoral artery, in the middle of the experiment, and then decapitated. Neither procedure caused any significant variation in the pressure. Still other experiments were done on the decapitated heads of freshly killed dogs. These recorded the same phenomenon of a rapidly forming negative pressure.

These experiments on the trachea of the hen and the sinus of the dog demonstrate another mechanism for the removal of air and the development of negative pressure. It is essentially a piston-cylinder action motivated by ciliary power, the masses of mucus acting as the pistons. If this factor is acting in the development of postoperative atelectasis, we would have the explanation for the failure of the soft mucus to slide into the area of negative pressure and for the seeming inaction of the cilia. When the cilia have pushed the masses of mucus up a bronchus as far as they are able, against atmospheric pressure, the mass of mucus becomes stalled. This explains the fact that very soft mucus can seemingly act like a cork; the cilia are holding it in position and attempting to push it further. The negative pressures of 200 to 400 mm of water, which have

actually been measured in cases of postoperative atelectasis, might readily be explained on the basis of a series of mucus pistons in tandem being pushed upward in the bronchial tree by ciliary action. This negative pressure could, theoretically, be produced entirely by ciliary action independent of absorption. The negative pressure is probably maintained entirely by ciliary power.* One can say that the effective power of the cilia equals the atmospheric pressure minus the pressure in the affected lobe and is the magnitude of the negative pressure. This pumping action of the ciliary mechanism is similar in principle to that of a mercury vacuum pump.

It was found in the experiments on the trachea of the hen that if the procedure were carried on for an hour or more the cilia continued to whittle away at the periphery of the mucus piston, gradually carrying it upward and depositing it at the end of the trachea. Theoretically, the cilia could remove the piston maintaining the negative pressure in atelectasis also, if given sufficient time, provided no more mucus formed. However, if production of excessive mucus should continue, then the atelectasis might be maintained.

We have spoken of absorption of air from the sinuses as though it were the usual thing for the ostium to be obstructed while the cavity is still full of air. It is more likely that in conditions resulting in vacuum headache the air was initially displaced more or less completely by the first secretion which formed. It seems likely that the heavy mucus, which forms large masses toward the end of an attack of sinusitis, might produce a negative pressure in a manner similar to that in the experimental dogs. This could very readily be the explanation for the clinical condition known as vacuum headache.

This last suggests another possibility for the rapid removal of air from the lobe of a lung in a surgical patient during operation. It is quite possible that the air is largely displaced by a secretion and that a negative pressure soon follows due to ciliary action.

The negative pressure which occurs in the middle ear might very likely be on the basis of ciliary action moving pistons of mucus down the eustachian tube. Attempts to actually demonstrate this in the eustachian tubes of beef animals killed at the packing plant

*There is another mechanism possible which should be mentioned. If the occluding mucus piston occurs in a membranous bronchus, it is quite possible that the bronchus may collapse behind the piston, due to the negative pressure. If this were to happen, the apposing walls of the bronchus might adhere together because of the cohesiveness of the normal mucous film. In this case, the atelectasis might conceivably be maintained without the presence of an abnormally great quantity of mucus.

have failed repeatedly. I do not know why it is so difficult to demonstrate ciliary action in the eustachian tube on this animal. Microscopic examination reveals that the epithelium of the tube is well ciliated. Even if mucus were produced in the tube and carried down by ciliary action, it is conceivable that no air would be removed in this way because the mucus could be produced distal to the air. On the other hand, it is a little difficult to understand how a considerable negative pressure can be produced in the middle ear in less than half an hour by absorption alone. It is true that the mastoid cells roughly approach lung formation but the blood supply is normally meager, which is true, too, of the middle ear. Moreover, in comparison to the alveoli of the lung, the endothelial lining of the middle ear and eustachian tube is thick. Just how the air is removed from the middle ear in blockage of the eustachian tube remains an open question.

SUMMARY

The absorption of air from the anterior chamber of the eye, from the sinuses, from the ear and from obstructed portions of the lung follows the principles of well known laws of physics such as those governing diffusion, solubility, partial pressures and molecular combination. It seems that air can be completely absorbed from almost any space or tissue in the body, such as those just mentioned, as well as the peritoneum, pleural cavity and subcutaneous tissues, provided sufficient time is allowed and provided the space containing the air can collapse. If the space containing the air cannot collapse or can do so only partially, a negative pressure develops, depending upon the gas pressures present in the absorbing fluid.

In postoperative atelectasis, there are at least three mechanisms by which the air could be removed, namely (1) absorption, (2) displacement by secretion and (3) the pumping action of cilia and moving masses of mucus. The negative pressure in most cases of postoperative atelectasis is, in all probability, maintained solely by ciliary action. It is probable that the mechanism involved in the pumping action of the cilia and moving masses of mucus is the one which causes vacuum headache and possibly also negative pressure in the middle ear following otitis media.

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REFERENCES

1. Coryllos, P. N.: Postoperative Apneumatosis (Atelectasis) and Postoperative Pneumonia; Experimental Evidence, J. A. M. A. 93:98-99 (July) 1929.

2. Coryllos, P. N., and Birnbaum, G. L.: Bronchial Obstruction; Its Relation to Atelectasis, Bronchopneumonia and Lobar Pneumonia; Roentgenographic, Experimental and Clinical Study, Am. J. Roentgenol. 22:401-430 (Nov.) 1929.

3. Coryllos, P. N., and Birnbaum, G. L.: Circulation in Compressed, Atelectatic Lung, (Pneumothorax—Apneumatosis—Pneumonia), Arch. Surg. 19:1346-1424, Dec. (Pt. 2) 1929.

4. Habliston, Charles C.: Intrapleural Pressures in Massive Collapse of the Lung, Am. J. M. Sc. 176:837, 1928.

5. Henderson, Yandell: The Physiology of Atelectasis, J. A. M. A. 93:96-98, 1929.

6. Herbert, C.: Personal communication.

7. Hilding, A. C., and Hilding, David: The Volume of the Bronchial Tree at Various Levels and Its Possible Physiologic Significance, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 57:324 (June) 1948.

8. Hilding, A. C.: Some Further Experiments in Production of Negative Pressure in the Trachea and the Frontal Sinus by Ciliary Action, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 54:725-738 (Dec.) 1945.

9. Marshall, James M.: Postoperative Pulmonary Atelectasis, U. S. Nav. M. Bull. 42:601-606 (Mar.) 1944.

10. Schmidt, Herbert W., Mousel, Lloyd H., and Harrington, Stuart W.: Postoperative Atelectasis; Clinical Aspects and Review of Cases, J. A. M. A. 120:859-900 (Nov.) 1942.

11. Seybold, Wm. D.: Physiologic Disturbance Underlying the Development of Early Postoperative Atelectasis after Lobectomy, Surg. Clin. North America 28:871-888 (Aug.) 1948.

XXVIII

THE TRANSMISSION PROPERTIES OF THE STAPES

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In a preceding study[1] we dealt with the middle ear as a mechanism for sound transmission, but because of the method that was used the results were limited to the more peripheral portion of that mechanism: to the drum membrane and the two outer ossicles and their suspensory system. Now, by a somewhat different method, we are able to extend the discussion to the third ossicle, the stapes. Our problem specifically is the manner in which the stapes affects the sounds that it transmits inward to the cochlea.

These experiments, like the foregoing ones, made use of the cat as an experimental animal, and gave measurements of the electrical potentials produced in the cochlea during stimulation with sounds. The animals were deeply anesthetized with diallylbarbituric acid by intraperitoneal injection, and usually were further immobilized with curare. They were maintained at a constant physiological level by artificial respiration.

The preparatory procedure was as follows. With special care to avoid any injury to the stapes, the auditory bulla was opened and the incudostapedial joint was severed, and then the drum membrane and the two outer ossicles were removed. Two sound tubes were applied to the ear, one over the oval window and the other over the round window. The one over the oval window surrounded the stapes but was adjusted to make no contact with it. These tubes led from separate loud-speakers which emitted tones that were of the same frequency but that could be varied independently in other respects. The arrangement for this purpose included an audio-oscillator that fed into a divider network, and thereafter two sep-

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arate channels of attenuators and amplifiers. In addition, one of the channels contained a phase shifter. By these controls the tones could be given any desired intensity and phase relations.

Both sound tubes included acoustic probes, which consisted of a miniature condenser microphone fitted with a front cap and an extending probe tube. The probe tube entered the sound tube through its side wall and then ran concentrically with it to its termination. This probe apparatus gave an indication of the sound pressures existing at the ends of the sound tubes and hence at the two windows.

A needle electrode was placed as close as possible to the round window for the recording of cochlear potentials.

The sound tubes were applied over the windows with sufficient firmness to prevent any appreciable leakage of sound from one location to the other. This isolation of the sources was always checked by introducing sound into one tube at a time and recording from both probes.

The further procedure consisted of applying tones simultaneously at the oval and round windows and adjusting the intensity and phase relations so as to obtain a cancellation of cochlear potentials. This was most readily done by first setting the intensity in each channel separately to an amount producing some desired level of cochlear potential. Then after the two stimuli were combined it was only necessary to vary the phase in one channel to reduce the response to zero. Thereupon by means of the acoustic probes a reading was made of the intensities of the two stimuli and of their phase difference.

It is possible to show that the stimulation by way of the oval window and by way of the round window involves the same receptor cells and in the same pattern of activity.[2] The evidence is that once a minimum response is obtained it holds for any location of the electrode over the surface of the cochlear capsule. This fact signifies that the cancellation of response occurs at every locus along the basilar membrane and hence for each and every sensory cell. Also, as we have concluded earlier, the cancellation occurs when the basilar membrane is exposed to two sound pressures that at every locus are equal in magnitude and opposite in direction.

We have further concluded that under the condition of cancellation the sound waves entering the two windows have the same intensity and phase except as these are altered along the routes. An agreement of phase with respect to the exterior becomes a contrary

phase at the basilar membrane because the waves approach this membrane from opposite sides.

It is from the above considerations that we speak of our readings of intensity and phase as representing differences between the oval window and round window pathways. More specifically, we refer these differences to the windows themselves. Our reasons are as follows. The two pathways through the cochlea differ little in length, and hardly permit of much attenuation or phase change on this account. There are certain anatomical differences, to be sure. The oval window pathway traverses the perilymph of the scala vestibuli and then Reissner's membrane and the endolymph of the cochlear duct, whereas the round window pathway traverses only the perilymph of the scala tympani. However, Reissner's membrane is an exceedingly thin and yielding membrane. The endolymph probably has the same acoustic properties as the perilymph. Hence there is little reason to attribute to these anatomical features any special effect upon the transmitted sounds. They may have some effect, but we consider it slight in comparison with the effects of the windows. Their structural differences are obvious: the oval window contains the stapes, whereas the round window is covered simply by a thin membrane.

As will soon be brought out, there is further evidence within our results that reinforces the inference that by this method we are dealing with the acoustic properties of the stapes.

RESULTS

As reported previously,[3] the oval and round window routes vary somewhat in effectiveness. The sensitivity as shown in the cochlear potentials is a little higher when the stimuli are presented to the oval window. Figure 1 shows some of the results. The curves represent differences in the stimulus intensities necessary to produce a standard response, with positive values signifying a greater sensitivity by the oval window route. The solid-lined curve is for an ear in which the stapes was intact and the dashed curve for one in which only the footplate remained in the oval window.

It is clear that when the stapes is intact the oval window route is consistently the more sensitive, though only by small amounts. The differences in favor of this route are largest in the upper frequencies, at some points attaining as much as 9 db. When the head and crura of the stapes are removed, leaving only the footplate in the oval window, these differences are smaller. In the low frequencies they cease to show any certain trend, though in the high

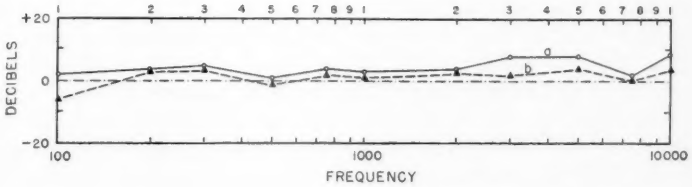

Fig. 1.—Sensitivity effects attributed to the stapes. Plotted are the differences in decibels between the stimulus intensities required at oval and round windows to produce a standard response of 10 microvolts, with positive values indicating a smaller requirement at the oval window and hence a greater sensitivity at this position. Curve *a* is for an ear in which the stapes was intact, and curve *b* is for an ear in which only the stapedial footplate remained.

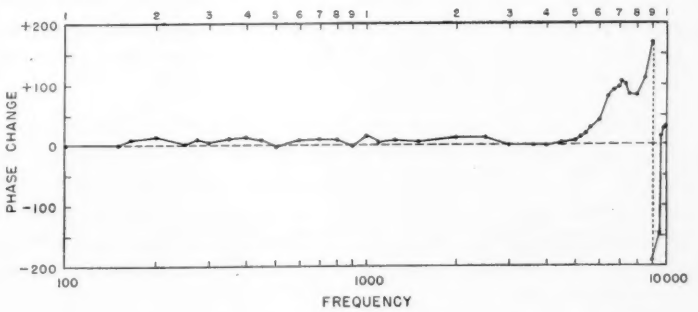

Fig. 2.—Phase effects attributed to the stapes. The curve shows the phase differences between tones presented simultaneously at oval and round windows and adjusted for zero response. Positive values represent a lag of phase and negative values an advance of phase as a result of the action of the stapes.

frequencies there is still a small but consistent difference in favor of the oval window route. This difference now does not exceed 4 db.

Some of our observations on phase differences between the oval and round window routes are shown in Fig. 2, for the condition in which the stapes was intact and its tendon remained attached. In this figure a positive phase change signifies that for a cancellation of response the tone presented to the oval window had to be advanced in phase relative to the tone at the round window. Or, according to our interpretation, a positive phase change signifies that the stapes in the oval window route introduced a phase lag into the transmitted sound.

The curve shows little phase variation over the lower portion of the frequency range up to 5000 cycles. At no point between 100 and 5000 cycles does the phase difference exceed 15°. The differences that do appear are consistently positive, however, indicating a small phase lag.

Beyond 5000 cycles the phase lag increases rapidly with frequency and attains a maximum at 9000 cycles. As a lag of phase that exceeds 180° becomes an advance of phase, the function is discontinuous here and suddenly appears as a negative phase. This negative or advancing phase grows rapidly less and the curve crosses the zero phase line at 9400 cycles. The discontinuity at 9000 cycles, which is virtually a crossing of the zero line from above downward, indicates a point of antiresonance, and the crossing from below upward at 9400 cycles indicates a point of resonance.

Results for another ear are shown in Fig. 3. Here the lower half of the frequency scale has been cut off, as it exhibits no new features. The solid-lined curve (curve *a*) shows the phase differences for the same condition as in the preceding figure, when the stapes was intact. The form of the function is the same as before, but in this ear the antiresonance is at 8800 cycles and the resonance is at 10,500 cycles.

This figure presents further the results of surgical modifications of the stapes. Curve *b* shows the phase function obtained after cutting the stapedius tendon. This curve rises more rapidly above the zero phase line than curve *a* does, and attains a maximum at 6900 cycles. It then appears as a negative phase and thereafter follows fairly closely the course of the former curve. Its antiresonance is at 6900 cycles and its resonance is at 11,000 cycles.

Then in this same ear the head and crura of the stapes were removed, leaving only the footplate in the oval window. The meas-

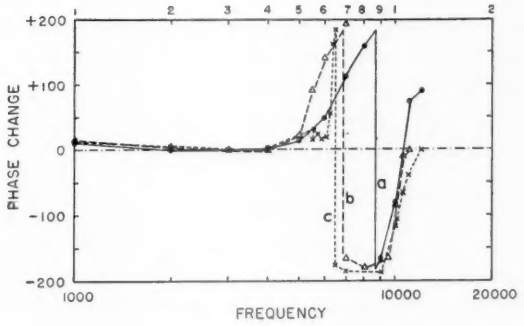

Fig. 3.—The effects on phase relations of surgical manipulations of the stapes. For curve *a* the stapes was intact, for curve *b* its tendon was severed, and for curve *c* its head and crura were removed.

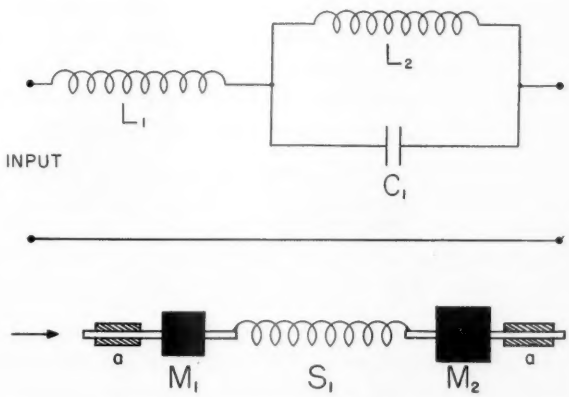

Fig. 4.—Electrical and mechanical "models" of the stapedial system. The diagram above gives an electrical circuit that exhibits the same form of phase response as shown in Fig. 2. The diagram below is its mechanical counterpart. In the electrical circuit, L_1 and L_2 are inductances and C_1 is a capacitance. In the mechanical system, M_1 and M_2 are masses and S_1 is a spring or compliance. The vibratory force is applied to M_1. In this system the connecting shafts are to be considered massless, and they move in bearings *a*, *a* with little or no friction.

urements then gave curve *c*. This curve, after some irregularities around 5600 cycles, undergoes a particularly rapid rise to a maximum phase lag at 6500 cycles. It becomes discontinuous like the others and then rises to cross the zero line. Its antiresonance is at 6500 cycles and its resonance is at 12,000 cycles.

During all these manipulations on the stapes the function in the low frequencies remained unaffected.

DISCUSSION

The observation of considerable changes in the forms of the phase curves in the high frequencies as a result of modifications of the stapes adds weight to our conclusion that the phase differences are mainly due to this ossicle in the oval window. It follows that these differences should disappear if we were able to eliminate this ossicle altogether. Unfortunately it is not possible to do so. Removal of the footplate opens the window and permits the escape of perilymph, thereby causing a serious and progressive impairment of the cochlear response.

Let us now attempt an analysis of the mechanical properties that are necessary to give rise to the phase changes shown. In this analysis, as in our earlier treatment of the middle ear mechanism, it is convenient to use an analogical method and first to ascertain the form of electrical circuit that exhibits the same properties.

Such a circuit must contain at least three reactive elements: two inductances and one capacitance. A possible arrangement is shown in the diagram at the top of Fig. 4. This circuit, when the proper values of the elements are chosen, will present an antiresonance at one frequency and a resonance at a higher frequency, as required.

The mechanical counterpart of this circuit is presented in the diagram at the bottom of this same figure. It consists of the two masses M_1 and M_2 and one compliance S_1 assembled in the manner shown.

The identification of these mechanical elements in the ear is rather uncertain. No doubt the mass M_1 includes the stapes and the compliance S_1 includes its suspensory ligament in the oval window. It is probable, however, that we need to lump with these parts certain reactive properties of other tissues within the cochlea itself. Some of the fluid just behind the stapes will move strictly with this structure, thereby adding to its effective mass. Also, this fluid is under the elastic restraint of the cochlear capsule as a whole and some addition to S_1 will result. The mass M_2 is probably contributed by

the cochlear contents in so far as these are set in motion by the stapedial action.

It is necessary to bear in mind here that we are dealing with differences between oval and round windows. The whole elasticity and mass of the cochlear contents are not reflected upon the oval window, but only a part, namely, that part involved in the stimulation by this route and not by the other. This part is reflected, we believe, because in the high frequencies with which we are concerned the footplate is a more effective means of setting the cochlear fluid in motion than the membrane of the round window. That this is so is attested by the observation that for these frequencies the sensitivity is a few decibels greater when sounds are applied at the oval window than when applied at the round window.

The surgical manipulations that we are able to make at the oval window evidently cause only a quantitative change in the reactive elements, and do not alter the general picture. Cutting the stapedius tendon reduces the stiffness of the system, which means that the compliance is increased. The effect is seen as a reduction of the frequency of antiresonance. Removal of the mass of the head and crura of the stapes likewise reduces the frequency of antiresonance, though only slightly. Unexpectedly, these measures affect the resonance frequency relatively little and in a contrary direction.

For the ears represented here the frictional forces to which the mechanical system is subjected are particularly small, as indicated by the rapid changes in the phase curves. Especially is this true when only the footplate is present. In certain other ears included in the study the frictional resistance evidently was somewhat larger, as the curves at antiresonance were less abrupt. In any case, the resistance seems to be relatively unimportant among the conditions governing the stapedial action, and we have left it out of account in drawing up the representative schemes of Fig. 4.

SUMMARY AND CONCLUSIONS

The experiments show that in the cat, when the drum membrane and outer ossicles are removed, the oval window and round window routes to the cochlea present differences both of sensitivity and phase. These differences, we have reason to believe, are mainly the result of the presence of the stapes in the oval window.

The effects of this ossicle on sensitivity are slight, not exceeding 9 db when the stapes is intact and not exceeding 4 db when only the footplate remains, and in either case are most noticeable in the high frequencies.

The effects on phase are small over the main course of the frequency scale, and then become marked for the high frequencies. Above 5000 cycles a phase lag enters and mounts rapidly to a maximum, whereupon it changes in discontinuous fashion to a phase advance. Thereafter the curve approaches and crosses the zero phase line. The discontinuity represents a point of antiresonance and the crossing from advancing to lagging phase represents a point of resonance. The locations of these points vary somewhat as the stapes is modified by the removal of its more peripheral parts.

An attempt is made to analyze the action of the stapes in the process of conduction by the use of electrical and mechanical "models."

It is of interest that the small changes that are produced by the stapes over the low-frequency range are consistently in the direction of a phase lag, whereas the changes produced in the low frequencies by the other parts of the middle ear, as our previous study showed, are mainly a phase advance. The result of adding the stapedial function to this other one is to bring the over-all response for the range from 200 to 1000 cycles closer to zero phase and hence to improve the mechanical tuning of the ear in this range.

The stapes is of some small service also for the medium high frequencies, as shown by the sensitivity data. For the uppermost frequencies, however, it largely adds complexities to the mechanical vibratory characteristics: its effect is disadvantageous in one region and advantageous in another.

We are indebted to the Bell Telephone Laboratories and the Western Electric Company for the loan of calibrated microphones used in this research.

PRINCETON UNIVERSITY.

REFERENCES

1. Wever, E. G., and Lawrence, M.: The Transmission Properties of the Middle Ear, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 59:5 (Mar.) 1950.

2. Wever, E. G., and Lawrence, M.: The Acoustic Pathways to the Cochlea, J. Acoust. Soc. Amer. 22:4 (July) 1950.

3. Wever, E. G., Lawrence, M., and Smith, K. R.: The Middle Ear in Sound Conduction, Arch. Otolaryng. 48:19-35, 1948.

XXIX

A CLINICAL STUDY OF AUDITORY DAMAGE FOLLOWING BLOWS TO THE HEAD

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Deafness may follow a blow to the head. Patients suffering from such a deafness may or may not have had a fracture of the temporal bone. A number of investigators have described the changes in the middle and inner ear in patients having had a recent fracture of the temporal bone. There are also several reports concerning the inner ear findings in patients having had a hearing loss following a head blow without temporal bone fracture. A review of these contributions leaves considerable doubt as to the exact physiopathological basis for the inner ear type deafness which occurs in some of these patients.*

The present paper deals with the clinical aspects of deafness following head trauma. The inner ear pathology as found in cats subjected to head blows will be the subject of a future report.

Several different types of auditory damage following head injury are recognized. The following simple classification is useful.

1. Hearing loss associated with longitudinal fracture of the temporal bone.

2. Hearing loss associated with transverse fracture of the temporal bone.

3. Hearing loss without evidence of temporal bone fracture.

Group 3 may be divided into: (a) Those cases with skull fracture not involving the temporal bone, and (b) those cases with no demonstrable skull fracture. The nature of the damage to the audi-

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*The term "inner ear deafness" will be used in this paper to indicate any hearing loss due to pathology other than that attributable to the conduction system. Commonly used synonyms are "nerve deafness" and "perception deafness."

tory organ in either case is probably the same so that this distinction is unnecessary.

Alexander and Scholl[1] found demonstrable hearing losses in 33% of 551 cases of head injury (commotio cerebri, 15%; basal skull fracture, 80%). Disturbances in cochlear and vestibular function comprised the largest group of objectively demonstrable late changes following head injury.

1. *Longitudinal Fracture of the Temporal Bone.* The typical longitudinal fracture exists as a linear break parallel and adjacent to the anterior margin of the pyramid in the floor of the middle cranial fossa. It extends medially to the region of the gasserian ganglion and laterally to the roof of the middle ear or into the mastoid cells. When the fracture line extends into the squamous portion of the temporal bone, it is easily seen on the roentgenogram. The mastoid air cell system is frequently less radiolucent because of the presence of blood or cerebrospinal fluid. According to Fischer and Wolfsen[2] about 80% of temporal bone fractures are of the longitudinal type. There is no known special type of head blow which has the selective quality of producing the longitudinal fracture of the temporal bone.

There is commonly a fracture through the superior part of the annulus tympanicus and a tear in the adjacent tympanic membrane. In such cases there is bleeding from the ear which may continue for a day or more. When the bleeding is slight one may find only clotted or dried blood in the external auditory canal. Ruttin[3] found bleeding from the ear in 29 of 50 cases of skull fracture. A step-like deformity in the external auditory canal may occur when the fracture extends through it. Grove[4] found the external auditory canal involved in six of 150 cases of skull fracture.

In Ulrich's experience[5] there was facial weakness or paralysis in less than 25% of cases of longitudinal fracture of the temporal bone. As a rule the facial paralysis is temporary. Cerebrospinal otorrhea is seen in a small percentage of cases and subsides in a few days.

Escher[6] has described three types of hearing loss occurring in patients with longitudinal fracture of the temporal bone. They are: (1) Hearing loss of the conduction type with the greatest deafness in the low tone range, (2) a combined conduction and inner ear type deafness with the greatest loss in the high tone area, and (3) a combined conduction and inner type deafness with the entire range of frequencies depressed.

Our cases do not fall categorically into such a classification. The occurrence of conduction deafness alone in the fractured ear is uncommon. Some degree of inner ear deafness is almost always associated with the conduction loss. In mild cases there may be only a 4096 (C_5) dysfunction whereas in severe cases there may be a marked hearing loss involving all the frequencies.

Our findings agree with those of Ruttin[3] in that the hearing loss in the ear opposite the fracture is predominantly of the inner ear type.

The audiograms and clinical histories of eight selected cases of longitudinal fracture of the temporal bone follow:

CASE 1.—F. C. On June 7, 1936, this 11-year-old boy was struct by a streetcar and momentarily lost consciousness. There was bleeding from the left ear.

Examination revealed a normal appearing right tympanic membrane. The left tympanic membrane was bluish in color, and there was a tear in its posterior superior quadrant. The Rinne test was negative in the left ear, and the Weber test lateralized to the left. No nystagmus was noted. Roentgenograms demonstrated a linear longitudinal fracture of the left temporal bone in the region of the sinodural angle.

An audiogram taken two weeks later is seen in Fig. 2.

Comment: Two weeks following injury the hearing had returned almost to normal for all tones in the fractured ear. The opposite ear showed a marked high-tone depression. It is doubtful that this high-tone deafness was present in this 11-year-old boy before the injury.

CASE 2.—A.K. On November 28, 1938, this 28-year-old man fell from a moving automobile, striking his head on the pavement. He was unconscious for several minutes. There was no bleeding from the ears. For several days he experienced drowsiness and lethargy; there was dizziness with sudden movements of the head. Seven days after the injury he first noticed deafness in the left ear. He stated that the ear felt as if it had water in it. There was no tinnitus.

He was first seen by us on the eleventh day following injury. The left tympanic membrane was intact but pinkish-gray in color and bulging slightly. There was no bluish color to the drum membrane. There was no blood in the external auditory canal. Roentgenograms of the skull demonstrated a linear longitudinal fracture

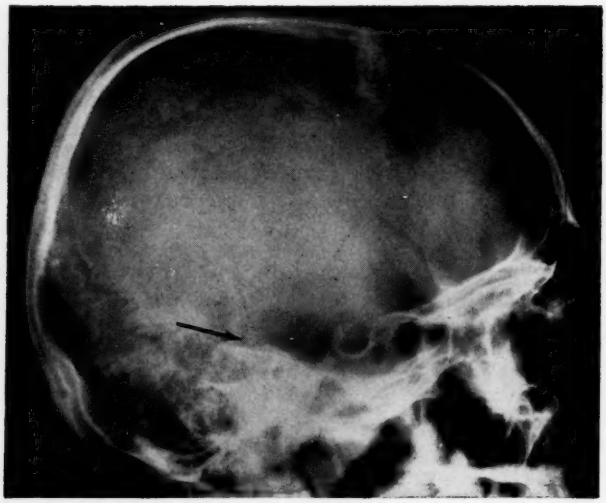

Fig. 1, Case 3.—R. P. (7-30-38) Linear longitudinal fracture of the left temporal bone extending into the pars squamosa (arrow).

of the temporal bone extending into the mastoid cells. On the twenty-second day the left tympanic membrane still appeared dull. Hearing was much improved. Audiograms taken on the eleventh and twenty-second days are shown in Fig. 2.

Comment: In this case the longitudinal fracture extended into the mastoid bone. The fracture probably did not involve the annulus tympanicus or drum membrane because there was no bleeding from the ear. The seven-day latent period before deafness was noticed by the patient may have been due to masking by the more severe symptoms of cerebral concussion. There was improvement in hearing for low tones between the eleventh and twenty-second days.

CASE 3.—R. P. On July 30, 1938, this 18-year-old boy fell from a moving automobile and received a severe blow to the left parieto-occipital area. He was unconscious for five minutes. There was bleeding from the left ear for three days and his hearing was poor in that ear. Also, he had a left facial weakness which improved somewhat over a period of two months. Roentgenograms showed a linear longitudinal fracture extending from the middle of the left parietal bone downward through the base of the petrous pyramid posterior to the external auditory canal (Fig. 1). Examination re-

vealed the right tympanic membrane to be normal. The left external auditory canal was filled with blood. The Rinne test was positive on the right and negative on the left. The Weber test showed lateralization to the left.

Six weeks later the left tympanic membrane was bluish in color and retracted, and hearing was still poor.

Eight weeks following injury the drum had lost its bluish color but was markedly retracted. Hearing was considerably improved. (See audiograms taken at six and eight weeks after injury in Fig. 2.) There is a combined conduction and inner ear type deafness in the fractured ear.

Comment: In this case the fracture involved the annulus tympanicus and drum membrane. Resolution of the middle ear process apparently occurred between the sixth and eighth week because the hearing for low tones improved during this period. The high-tone hearing losses in both Cases 2 and 3 showed no improvement during the time observed. In both cases the opposite ear had a slight high-tone depression.

CASE 4.—J. W. On June 30, 1939, this 23-year-old student was found unconscious in an alley. He was bleeding from the left ear. He regained consciousness in about four hours.

Examination revealed a left facial weakness. The left external auditory canal was filled with blood. The fracture could not be demonstrated on roentgenograms.

The audiogram seen in Fig. 2 was taken two months after injury. There was a mild bilateral high-tone loss. The facial weakness had disappeared.

Comment: Because of the young ages of patients in Cases 2, 3 and 4 it appears likely that they had normal hearing before injury. In no case was there a known deafness before injury.

CASE 5.—N. K. On October 9, 1946, this 59-year-old steel worker received a blow to the left parietal region by a falling piece of lumber. He was unconscious for five minutes. When he recovered he noted headache, dizziness, deafness, and tinnitus. There was no bleeding from the ears. Roentgenograms revealed a linear longitudinal fracture involving the left temporal and parietal bones. When he was first seen by us seven months later, he still complained of deafness and tinnitus. The tympanic membranes appeared normal. Positional nystagmus tests and cold caloric tests gave normal results. The audiogram is seen in Fig. 2. The audiogram showed a

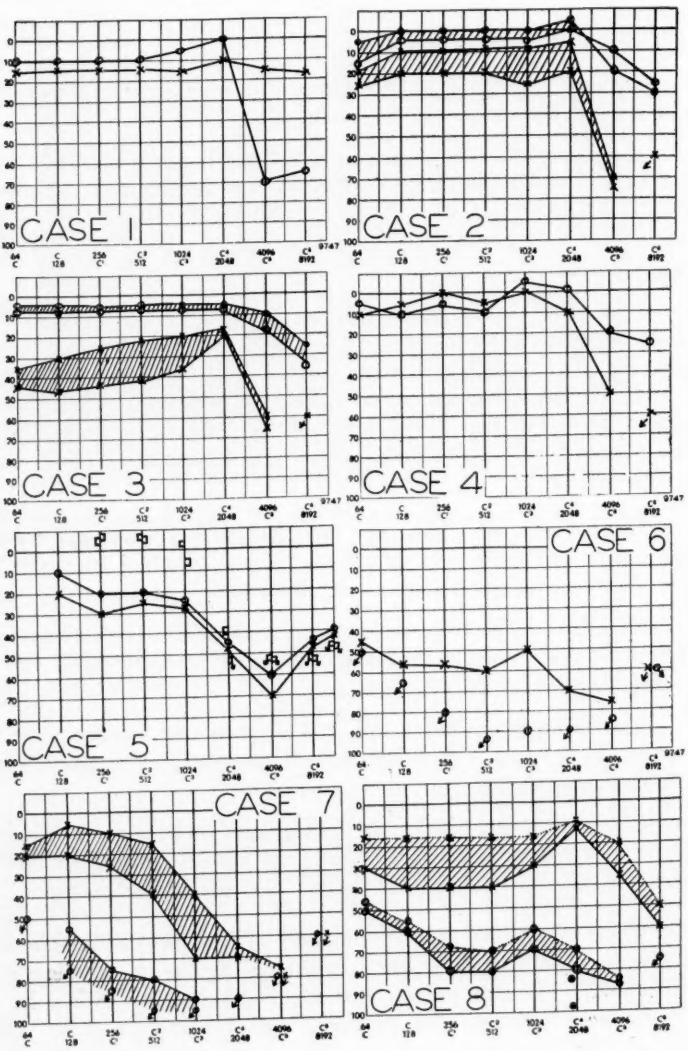

Fig. 2.

Fig. 2.—Audiograms of patients sustaining a longitudinal fracture of the temporal bone.

Air conduction: right=O, left=x; bone conduction: right], left [. Arrows indicate tone was not heard at maximum intensity.

Case 1.—F. C. (6-22-36) Two weeks after a longitudinal fracture of the left temporal bone.

Case 2.—A. K. The hearing improvement between the eleventh and twenty-second days following a longitudinal fracture of the left temporal bone.

Case 3.—R. P. Audiometric improvement between the sixth and eighth week following longitudinal fracture of left temporal bone.

Case 4.—J. W. (8-30-39) Two months following longitudinal fracture of left temporal bone. Weber midline. Rinne positive bilaterally (512 fork).

Case 5.—N. K. (4-26-47) Seven months following longitudinal fracture of the left temporal bone. Fifty-decibel thermal noise masking used for bone conduction testing.

Case 6.—W. K. (11-23-38) Four days following longitudinal fracture of the right temporal bone.

Case 7.—A. F. Hearing improvement between the third day and fifth month following a longitudinal fracture of the right temporal bone. The right ear was tested with 60-cycle masking of about 70 db intensity in the left ear.

Case 8.—H. P. Improvement between the 3rd and 10th week following a longitudinal fracture of the right temporal bone. The right ear was tested with a 60-cycle tone of about 70 db intensity in the left ear.

bilateral hearing loss of the inner ear type progressive from the lower to the higher tones and most severe at the 4096 frequency.

Comment: This type of deafness is characteristic of so-called boilermaker's deafness and therefore in view of the patient's occupation it is not possible to state whether the hearing loss was incurred at the time of injury. We do know, however, that he first noted deafness and tinnitus immediately on recovering consciousness following the injury. The headache and dizziness are symptoms characteristic of postcerebral concussion syndrome.

CASE 6.—W. K. On November 19, 1938, this 44-year-old male pedestrian was injured by a hit-and-run driver. He was unconscious for several hours. There was bleeding from the right ear.

Examination four days later revealed the right external auditory canal to be filled with a blood clot. The tympanic membrane appeared intact but bluish in color. The left tympanic membrane appeared normal. The Rinne test (256 fork) was negative on the right (left ear masked) and positive on the left. The Weber test lateralized to the right. The only tone heard in the right ear by air conduction audiometry was the 1024 frequency. This tone was still heard when the Barany noise box was placed in the left ear. The left ear showed a hearing loss of 50 db for low frequencies and becoming more severe in the higher range. The audiogram taken on the fourth day is seen in Fig. 2.

Comment: The hemorrhage from the right ear and the negative Rinne strongly suggest that there was a longitudinal fracture of the temporal bone on that side. The profound hearing loss in that ear could not be due to the conduction lesion alone. The positive Rinne and the normal tympanic membrane on the left side indicate that the deafness in the left ear is predominantly of the inner ear type.

CASE 7.—A. F. This 57-year-old man was struck by an automobile on August 12, 1937. He did not lose consciousness. He did not notice bleeding from his ears. He noted deafness in both ears immediately following the accident. He stated that the hearing became progressively worse over the next three days. He also had a bilateral tinnitus, more severe in the left ear. He felt slightly dizzy at times and had a mild frontal headache.

He was first seen by us on the third day following the injury. The hearing was depressed bilaterally. He could hear only a loud shout into the right ear with the left ear masked by a Barany noise box. The right tympanic membrane was intact and dull grayish

blue in color. There was a slight hyperemia of the left tympanic membrane. A crust of dried blood was seen in the right external auditory canal. There was a sustained spontaneous horizontal nystagmus to the left. The cold caloric response (10 cc of 78° F. water) was normal in the left ear and absent in the right ear.

Roentgenograms revealed an occipital skull fracture; however, a fracture in the temporal bone could not be definitely seen. The right hematotympanum and the dried blood in the right external auditory canal strongly suggest that there was a longitudinal fracture of the right temporal bone.

Five months later the hearing had improved. The cold caloric response was present but diminished in the right ear. Audiograms taken three days following injury and again five months later are shown in Fig. 2. Only the lower tones are heard in the right ear at very high intensities with masking. In the left ear hearing acuity diminishes rapidly from low to high tones, with no responses above the 4096 frequency.

CASE 8.—H. P. A boy, aged 9, was accidentally struck in the right temporal region by a baseball bat on July 20, 1933. He did not lose consciousness but was stuporous for several hours. There was no bleeding from the ears. He immediately complained of deafness.

When examined in this clinic three days later he had a spontaneous nystagmus to the left. The Weber test did not lateralize. At this time he could not hear a loud shout into the right ear when the left was masked with a noise box. There was dried blood obscuring a portion of the right tympanic membrane. Roentgenograms showed the right mastoid cells to be clouded; however, no fracture could be demonstrated. There was a diminished vestibular response following turning to the right. The left tympanic membrane appeared normal. Audiograms taken three weeks following the injury and again two and a half months later are seen in Fig. 2. In the right ear the audiogram is essentially flat with losses between 50 and 90 db. In the left ear there is moderate deafness for low tones with a more severe drop for very high tones. Three weeks following injury he could understand words shouted into the right ear with the left ear masked with the Barany noise box, and the right tympanic membrane was intact.

Three years later the cold caloric test gave a normal response in the left ear but no response to ice water in the right.

An audiogram taken five years after injury (July 27, 1938) revealed no further change in hearing acuity. The left ear was masked with a 60-cycle tone while testing the right ear.

Comment: A conduction lesion is present in Cases 5, 6, 7 and 8 due to a fracture through the middle ear area. It is obvious, however, that a superimposed inner ear type lesion is contributing importantly to the hearing losses in these patients.

The prognosis for recovery of hearing in cases of longitudinal fracture of the temporal bone is generally favorable. Conduction type losses are always partially reversible. There is much less recovery from the inner ear (perceptive) type deafness (Ulrich,[5] Grove,[4] Brunner[7]).

Alexander and Scholl,[1] Grove,[4] and others have observed certain cases in which the hearing has continued to deteriorate for several months following a head injury. This has not occurred in our cases.

Pathology. The longitudinal fracture by definition does not involve the labyrinthine capsule. The ossicles are embedded in blood. Sakai[8] found fractures of the ossicles in a number of cases; however, Ulrich[5] and Barnick[9] have not been able to confirm this. Rupture of the articular capsules and subluxation of the incudostapedial joint are seen in the most severe cases (Kelemen[10]). The annular ligament may be torn. Ulrich found damage to the ossicular ligaments in five of 18 cases. He found the tensor tympani muscle or its tendon injured in seven cases; however, the stapedial muscle was always spared.

The nature of the lesion which is responsible for the inner ear type deafness is not fully understood. It is known that there may be hemorrhages in the internal auditory meatus, perilymphatic spaces, cochlear aqueduct and bony nerve canals in severely injured patients coming to autopsy. Ulrich saw gross hemorrhage in the cochlear aqueduct in 11 of 18 cases. Hemorrhage was most commonly found in the scala tympani, especially in the spiral ligament near the round window by Sakai, Ulrich, and others. Hemorrhage into the utricle, saccule, facial nerve, fallopian canal and spiral ganglion have been described (Sakai) but are uncommon. It is not known whether blood reaches these structures by diapedesis through a damaged vessel wall or by actual rupture of the vessel wall. Reissner's membrane is rarely injured.

In the average clinical case there is probably less severe middle and inner ear trauma than these descriptions indicate.

Other factors to be considered in explaining the inner ear deafness are direct damage to the hair cells by the traveling pressure wave, isolated cochlear fractures, and damage to the higher centers. (See discussion on inner ear concussion.)

Nassulphis[11] has examined histologically a number of fractured temporal bones and believes that mild hemorrhages may be absorbed completely. More severe hemorrhages, he states, may become organized and cause varying degrees of permanent hearing loss.

2. *Transverse Fracture of the Temporal Bone.* The transverse fracture occurs perpendicular to the long axis of the petrous pyramid. Axial and Stenvers' views of the skull demonstrate the fracture in about 50% of the patients (Grove[4]). The fracture often involves the facial canal at the geniculate ganglion. In this region the facial nerve does not lend itself well to surgical treatment. Facial paralysis occurs in over 50% of the cases and is often permanent.

There is rarely bleeding from the ear. Hematotympanum is a frequent finding. The drum membrane is dark blue at first, later becoming light blue and then rose-red before complete resolution occurs. Resolution occurs usually in seven to 14 days (Ruttin[3]).

Cerebrospinal fluid may fill the middle ear, in which case the condition is termed liquor tympanum (Voss[12]). There may be drainage of cerebrospinal fluid from the ear. Brunner, Voss, and Ruttin believe that cerebrospinal otorrhea is more common in transverse fracture than longitudinal fracture. In some patients suffering from a basal skull fracture the cerebrospinal fluid drains through the eustachian tube into the nasopharynx (Ecker[13]). The transverse fracture usually traverses the vestibule and causes extensive destruction of the inner ear structures with complete loss of cochlear and vestibular function. The patient suffers from extreme vertigo associated with nausea and vomiting which subsides gradually in one to four weeks. For several weeks there is falling toward the side of the lesion and spontaneous nystagmus toward the opposite side.

Some cases of incomplete loss of auditory or vestibular function following transverse fracture of the temporal bone have been reported. This brings up the question of the incidence of isolated or incomplete fracture of the labyrinthine capsule. Klingenberg[14] reported two cases in which vestibular function was preserved and cochlear function lost. In both cases fracture lines were seen on roentgenograms to extend through the pyramids into the internal auditory meatuses. De Kleyn and Stenvers,[15] Nager[16] and Hofmann[17] had similar cases. Schlittler[18] described a case in which histological examination revealed two small isolated fractures of the bony lab-

yrinthine capsule of the semicircular canal and one in the vestibule. Injury had taken place 16 years previously. Corti's organ and the spiral ganglion were completely degenerated.

Several clinical histories and audiograms of patients sustaining a transverse fracture of the temporal bone follow:

CASE 9.—F. S. In November, 1938, this 39-year-old male musician suffered multiple injuries when struck by an elevated train. He was unconscious for two hours. There was a slight hemorrhage from the right ear. He was very dizzy and vomited repeatedly for three days, and noticed that he could not hear with the right ear. The dizziness gradually subsided.

He was first seen by us two months after the injury. He complained of dizziness on sudden movements of the head. Examination revealed an absent cold caloric response in the right ear (5 cc ice water) and a normal response in the left (5 cc at 80° F.). There was a sustained spontaneous nystagmus to the left in all positions of the head. The drum membranes appeared normal. Auditory function tests showed normal hearing in the left ear but total deafness in the right. With the Barany noise box in the left ear he could not hear a loud shout into the right ear. Five and one-half years following the accident the vestibular symptoms were less severe, but the audiometric findings were unchanged. The audiogram is seen in Fig. 4.

CASE 10.—This 31-year-old physician was first seen on June 6, 1941. He stated that two years previously he had incurred a skull fracture in an automobile accident and was unconscious for five days. He had then suffered from intense vertigo for several weeks which gradually subsided. He also had a profound hearing loss since that time. There was no bleeding from the ears at any time. He stated that he still had a tendency to fall to the right.

Examination demonstrated normal appearing tympanic membranes. The cold caloric response was absent in the right ear (5 cc ice water) and normal in the left. He could not understand words shouted into the right ear when the left was masked with a Barany noise box. The skull roentgenograms demonstrated a transverse fracture across the right petrous pyramid (see Fig. 3). The audiogram (Fig. 4) shows a completely deaf right ear and a mild dip in the left ear.

CASE 11.—S. C. At the age of 42, in 1926, while crossing a street the patient was struck by an automobile and suffered severe injuries. He noted deafness and tinnitus in the right ear from that date. He experienced vertigo at that time. Two years later he

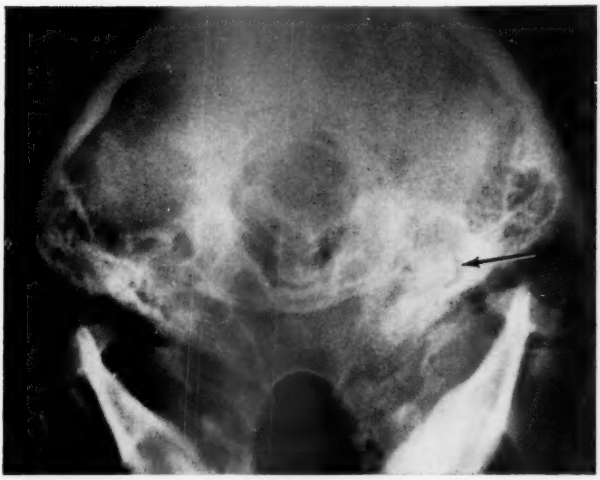

Fig. 3, Case 10.—Roentgenogram showing a transverse fracture of the right petrous pyramid (arrow).

began having grand mal seizures. He fell during such a seizure in 1943 and sustained a basal skull fracture. Following this second injury he noted deafness and tinnitus in the left ear.

Examination 13 years after the first injury (1939) revealed absent cold caloric response (5 cc ice water) in the right ear and normal response in the left (5 cc at 80° F.). The tympanic membranes appeared normal. Hearing was absent in the right ear and diminished for high frequencies in the left (see audiogram, Fig. 4). The audiogram was essentially unchanged following the second injury.

Case 12.—P. G. This 41-year-old man stated in 1944 that 33 years previously he had fallen from a third-story porch. He was unconscious for ten days and upon regaining consciousness he was very dizzy for about a month. He had noticed deafness and tinnitus in the right ear which had persisted. He began having convulsive seizures six months later and these had continued one to four times per week. The present examination revealed normal appearing tympanic membranes. There was no spontaneous nystagmus. The cold caloric test gave a normal response in the left ear but no response to ice water in the right. With the Barany noise box in the left ear the patient could not understand numbers shouted directly into the

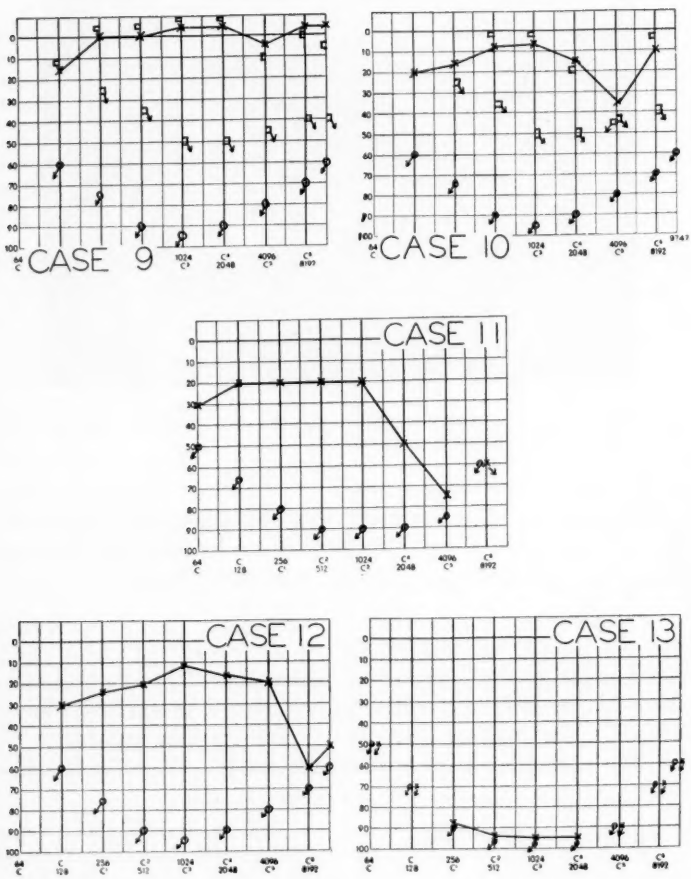

Fig. 4.

Fig. 4.—Audiograms in patients suffering a transverse fracture of the temporal bone. Vestibular and cochlear function are lost in the involved ear.

Air conduction: right=O, left=x; bone conduction: right], left [. The arrows indicate tones not heard at maximum intensity.

Case 9.—F. S. Audiogram taken two months following a transverse fracture of the right temporal bone.

Case 10.—W. R. Two years following a transverse fracture of the right temporal bone (see Fig. 3).

Case 11.—S. C. Auditory function 13 years after a transverse fracture of the right temporal bone.

Case 12.—P. G. Audiogram taken 32 years following a transverse fracture of the right temporal bone.

Case 13.—A. H. The auditory function 29 years following a severe head injury.

right ear. Roentgenograms revealed a post-traumatic fibrous degeneration of the skull involving the right parietal and occipital bones. The audiogram is seen in Fig. 4. There is total deafness in the right ear. A mild depression of the very low tones and a more marked loss for the very high tones is present in the opposite ear.

CASE 13.—A. H. The patient, a 53-year-old housewife, stated that she lost all hearing in an accident in which her skull was fractured 29 years before. After the injury she was unconscious for four days. She then noted deafness in both ears. She was dizzy for six months after the injury and still staggered somewhat when walking in the dark.

When first examined by us on September 17, 1947, she was found to have normal appearing tympanic membranes. The cold caloric test using 5 cc of water at 80° F. was normal in the left ear. There was no response to 10 cc of water at 70° F. in the right ear. The voice was high pitched and lacking in tonal quality, typical of a patient with profound deafness.

In the left ear she could hear several pure tones at high intensity as a "rumbling noise." There was no response to tones of maximum intensity in the right ear (see audiogram, Fig. 4).

Comment: One cannot state with certainty that there were transverse fractures in this case. Severe inner ear concussion without fracture might produce such a profound deafness. Complete loss of cochlear and vestibular function has been uniform in our patients with transverse fracture. The opposite ear was damaged in four of the five cases.

Pathology. The transverse fracture of the temporal bone usually traverses the vestibule and internal auditory meatus, but occasionally passes through the cochlea (Stenger[19]).

The tympanic membrane was intact in nine of 11 cases reported by Ulrich.[5]

Klestadt,[20] Mannasse,[21] and Hofmann[17] described an invagination of the middle ear mucosa into the vestibule through the fissure created by the fracture. Ulrich noted only granulation tissue in these fissures and a tendency to connective tissue closure. Healed round window tears are common.

Labyrinthine fractures are partially healed by new bone formation in one month. New borne forms in the region of the endosteum and periosteum whereas the enchondral areas are healed by connective tissue (Perlman[22]). Mayer,[23] Bast[24] and Nager[25] have

found that ossification processes in the enchondral bone are completed normally at the age of two. The extensive formation of connective tissue and new bone in the vestibule, cochlea, and canals has been described by Nager and Ulrich. An otitis media occurring many years following a transverse fracture may be complicated by meningitis because of partially healed or unhealed openings in the labyrinthine capsule leading to the perilymphatic spaces, internal auditory meatus, and the cochlear aqueduct (Ulrich).

3. *Deafness Following Head Trauma without Temporal Bone Fracture.* Deafness may follow a moderate blow to the head (Politzer,[26] Passow,[27] Schwartze[28]). The underlying pathology in these cases is not well understood.

The pathological findings described by Sakai, Barnick,[9] and Schonbauer and Brunner,[29] as well as the animal experiments of Stenger[19] and Brunner,[30] have demonstrated that hemorrhages may occur in the inner ear without fracture. The reports of Wittmaack[31] Theodore,[32] and Nassulphis[11] indicate that there may be severe degenerative changes in Corti's organ following head injury without fracture.

Histological studies are few because these patients do not die of their injuries. Uffenorde[33] and Ulrich have questioned the existence of direct end organ damage from a head blow. Ulrich considered the possibility of damage to the cochlear nerve by pulling or tearing in the internal auditory meatus.

It has been stated that the temporary depression in hearing following a head blow without fracture was due to loosening of the neurons or a reversible biochemical change in the nerve cell protoplasm. It is doubtful whether such changes can be proven by present histological methods.

Clinicians agree that deafness more frequently follows an occipital blow than blows to other parts of the head (Escher, Wittmaack, Koch,[34] Voss[12]).

The hearing in the ear opposite that of a longitudinal or transverse fracture frequently shows an inner ear type deafness in the 3000 to 8000 frequency range. This has been referred to as the C_5 or 4096 dip. This localized hearing loss must be considered as being due to concussion of the inner ear.

Concussion deafness in the ear opposite to a longitudinal fracture is illustrated in four cases shown in Fig. 5. In this figure hearing curves in four ears opposite transverse fractures are also demonstrated.

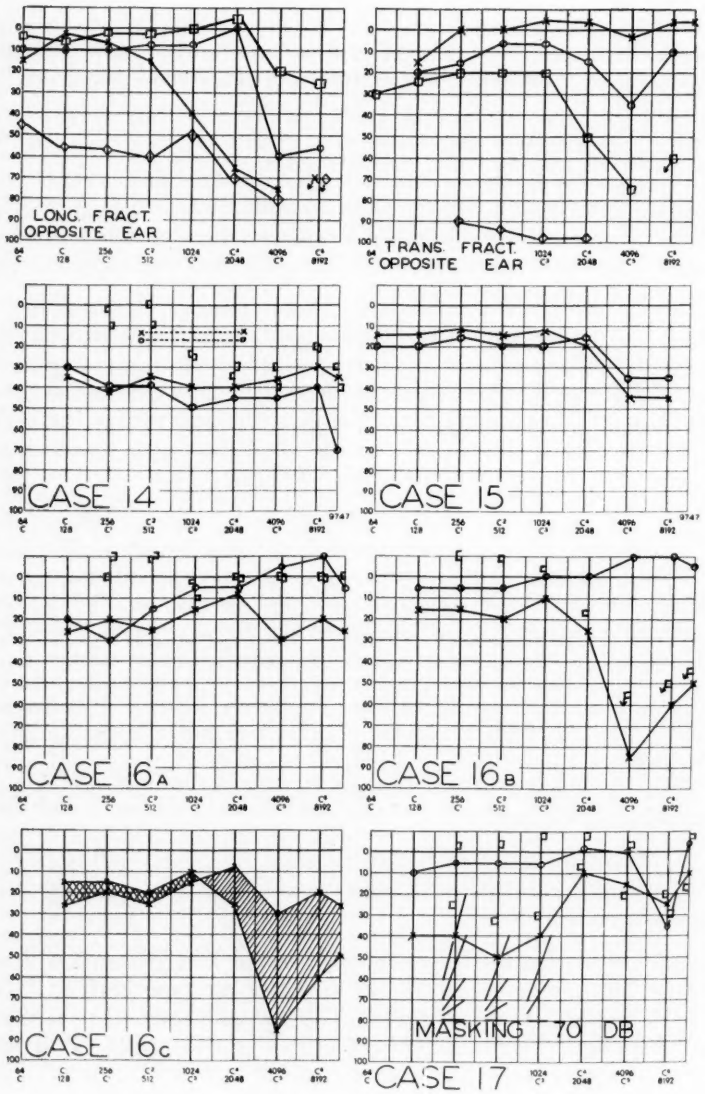
LONG. FRACT.
OPPOSITE EAR
TRANS. FRACT.
OPPOSITE EAR
CASE 14
CASE 15
CASE 16A
CASE 16B
CASE 16C
MASKING 70 DB
CASE 17

Fig. 5.

Fig. 5.—Auditory function in ears in which the temporal bone was not fractured (inner ear concussion).

Upper left: Auditory function in 4 separate ears. In each case there was a longitudinal fracture of the contralateral temporal bone.

Upper right: Auditory function in 4 ears. In each case there was a transverse fracture of the contralateral temporal bone.

Case 14.—Z. S. Auditory acuity six weeks following an occipital skull fracture without temporal bone fracture. The broken lines indicate hearing acuity by the Spondee word test. (Psychogenic deafness?)

Case 15.—G. H. Audiogram eight weeks after a head blow. No demonstrable skull fracture.

Case 16.—(*a*) Age 9. Chronic left suppurative otitis media and mastoiditis with cholesteatoma. Auditory acuity before surgery.

(*b*) Auditory function two months following a left modified radical mastoidectomy.

(*c*) Composite audiogram showing the amount of hearing improvement for low tones (cross lines) and the decreased hearing for high tones (diagonal lines) which resulted from surgery.

Case 17.—J. V. Audiogram taken two months after a left parietal skull fracture. There was no evidence of temporal bone fracture. There is a low-tone nerve type deafness on the left. Recruitment is present at these frequencies. (Ménière's disease?)

The following patients suffered from hearing losses following head blows without clinical evidence of temporal bone fracture.

CASE 14.—Z. S. This 54-year-old housewife suffered an occipital skull fracture in an auto accident in August, 1948. She was unconscious for 45 minutes. There was no bleeding from the ears. For two weeks she noted dizziness when she moved her head suddenly. She also believed her visual and hearing acuity to be diminished. When she was first examined by us six weeks after injury the tympanic membranes appeared normal. The audiogram revealed a 30 to 50-db loss for all tones in both ears. An inconsistency was the comparatively slight loss on the Spondee word test (Fig. 5). The cold caloric tests showed normal function (5 cc at 80° F.) On postural tests there was a slight upward vertical nystagmus in several of the positions tested. Her vision was 20/100 + 2 (aided) in both eyes and the visual fields were slightly constricted.

One month later she stated that both her vision and hearing were improved. Visual acuity proved to be 20/30 + 2 in the right eye and 20/30 + 1 in the left. The audiogram and the speech test were unchanged from the first examination.

Eight months after the examination her vision was 20/30 (aided) in both eyes. Although she now believed her hearing to be normal the audiogram and Spondee test revealed no change from the findings in Fig. 5, which were recorded eight months before. The discrepancy between the pure tone test and word test remained.

Comment: The auditory findings in this case are not conclusive for inner ear concussion. The discrepancy between pure tones and speech tests, the uniform loss throughout the frequency scale, and the accompanying visual disturbance are suggestive of psychogenic deafness.

Patients suffering from the postconcussion syndrome are susceptible to functional disorders.

This patient (Case 14) is presented as a case of postconcussion deafness, possibly of psychogenic nature.

CASE 15.—G. H. This 47-year-old male office worker received a severe head blow in an auto accident on July 22, 1938. He was unconscious for three hours. There was no bleeding from the ears and roentgenograms of the skull failed to reveal a fracture. At the time of our examination eight weeks later, he complained of dizziness when lying on his left side. He noted no tinnitus or hearing loss. Examination revealed a horizontal nystagmus to the left lasting several minutes when the patient was placed on his left side.

When lying on his right side there was a vertical downward nystagmus. Results of the cold caloric tests were normal (5 cc at 80° F.). The tympanic membranes were normal. The neurological examination revealed nothing significant. The audiograms showed a mild depression for all frequencies in both ears being more severe above 2048.

Comment: The cause for the depression of hearing for the frequencies below 4096 is probably not related to the head blow. The loss for the 4096 and 8192 frequencies, however, is more characteristic of shock pulse damage. Without preconcussion audiograms this point cannot be proven.

CASE 16.—B. B. Since the age of six months this boy had an intermittently draining left ear. At the age of 3 his tonsils and adenoids were removed. When 6 years old he had three radium treatments to the nasopharynx for hypertrophied lymphoid tissue.

He was first seen by us on March 4, 1949, at the age of 9. There was a perforation in Shrapnell's membrane on the left side and a foul smelling discharge with a intact membrana propria indicating an attic cholesteatoma. Roentgenograms of the mastoid bones were suggestive of cholesteatoma on the left side.

An endaural radical mastoidectomy was performed on the left ear on April 13, 1949. The mastoid bone was found to be sclerotic. The thickened cortex was removed by mallet and gouge, the cell partitions were removed by means of the electrically driven bur. A cholesteatoma of the epitympanic space was removed and the area opened widely. The procedure was modified to preserve the ossicles and the pars tensa of the tympanic membrane. The audiograms before and after surgery indicate that the patient incurred a high-tone deafness as a result of the operation (Fig. 5).

Comment: This appears to be an example of a localized hearing defect resulting from the use of the mallet and gouge in performing a mastoidectomy. In our experience such cases are uncommon. In this connection it is noteworthy that Ruedi and Furrer[35] and Escher[6] produced temporary hearing losses for the 4096 frequency in human volunteers by reflecting the scalp over the mastoid and delivering mallet blows directly to the bone.

CASE 17.—J. V. This 25-year-old male student was struck on the occiput by a passing bus while he was standing on the street (November 4, 1948). He was unconscious for several hours. There was no bleeding from the ears. Roentgenograms revealed a left parietal skull fracture. He first noticed diminished hearing in the left

ear several days later when using the telephone receiver on that ear. He also complained of a mild, fleeting postural vertigo which persisted for several months. The vertigo appeared to be characteristic of the type seen in the postcerebral concussion syndrome.

He was first seen by us eight weeks following the accident. The tympanic membranes appeared normal. The caloric test using 5 cc of water at 80° F. in the right ear produced a nystagmus of 40 seconds' duration to the left. The same test produced no reaction in the left ear. Ten cubic centimeters of ice water in the left ear produced a nystagmus of 30 seconds' duration to the right. The Weber test lateralized to the right and the Rinne test was positive bilaterally (512 fork). Audiometric and recruitment tests are seen in Fig. 5. Three examinations at weekly intervals showed no change in the caloric, audiometric and recruitment tests.

Comment: The hearing loss in this case is unlike any of our other cases or the cases of Escher.[6] The possibility of an early Ménière's disease exists. Notice, however, that the patient was only 25 years of age and that he did not complain of fluctuations in hearing or of tinnitus. The vertigo was fleeting in character and precipitated only by postural changes.

We are unable to state whether this case represents inner ear concussion or a pre-existing inner ear disease.

Pathology. This type of deafness has been termed inner ear concussion, commotio labyrinthi and otitis interna vasomotoria by various authors.

There are a number of opinions regarding the underlying pathology. Schwartze in 1883 conjectured that such a hearing loss when temporary was due to transient hyperemia of the membranous labyrinth and that longer hyperemia caused connective tissue formation and permanent damage.

Brunner delivered head blows to guinea pigs and in histological preparations was able to see perivascular infiltrates in the region of the spiral vein and hemorrhage in the scala tympani and cochlear aqueduct. He believed these changes to be vasomotor in origin and termed it otitis interna vasomotoria.

Because of the frequency with which blood is seen in the internal auditory meatus, Politzer suggested that the cochlear nerve might frequently be lacerated or its fibers torn in the porus acousticus internus.

Stenger[19] and Linck[36] studied the problem by delivering head blows to rats. Their main findings were inner ear hemorrhages in

the vestibule and scala tympani, particularly in the region of the round window. Stenger also found petechial hemorrhages in the vestibular and cochlear nerves near the porus acousticus internus. In some animals there was degeneration of Corti's organ and the spiral ganglion.

Wittmaack believed that a traveling pressure wave in the labyrinthine fluids could directly injure the hair cells (commotio labyrinthi). His experiments[31] were performed by delivering shock pulses directly to the labyrinthine fluids of cats. This was done by displacing the stapes into the vestibule and connecting a water filled rubber tube to the oval window. Weights were then dropped on the water column to produce the shock pulse. He found localized injury to the organ of Corti and spiral ganglion. The injury was most severe in the middle coil, next the apical coil, and least severe in the basal coil. The corresponding cells of the spiral ganglion lost their Nissl granules, became vacuolated and were severely degenerated in two weeks. He also saw isolated fractures of the osseous spiral lamina, tearing of the basilar membrane, tearing of Reissner's membrane and of the sacculus and hemorrhage in the perilymphatic spaces.

Ruttin believed the pressure wave injured the end organ directly by loosening the head plate of Corti's pillars. Spira, Wittmaack, Stenger, and others have mentioned the possibility of loosening of the hair cells or molecular alteration in their protoplasm. Following a study of human temporal bones Ulrich concluded that stretching or tearing of the cochlear nerve was the most common histological feature. He denies the possibility of direct injury to the end organ by the pressure wave.

Oppenheim[37] and others have suggested that commotio cerebri or petechial hemorrhages of the brain may lead to significant hearing loss by injury to the cochlear nuclei or higher centers. Deafness has been reported in bilateral temporal lobe tumors; however, the clinicopathological evidence of true central deafness is very meager (Tobey[38]).

Several case reports of inner ear concussion are worth mentioning. Stenger examined the intact temporal bone in a case of fatal skull fracture. He discovered hemorrhages in the cochlear duct, macula sacculi, macula utriculi, cochlear nerve, cochlear aqueduct and spiral ganglion.

Nager investigated the temporal bone of a 64-year-old "deaf mute" who suffered a head injury at the age of 4. He found no evidence of fracture; however, the labyrinth was filled with con-

nective tissue and bone. Mannassee, Alexander,[39] and Hellmann[40] in similar cases found connective tissue and bone in the perilymphatic spaces as well as severe degeneration of Corti's organ, cochlear ganglion and cochlear nerve.

Theodore[32] discussed the histological findings in a case of head injury. He found degeneration of the organ of Corti and spiral ganglion in the basal coil with no evidence of hemorrhage.

Wittmaack[31] describes a case in which there was severe hearing loss following a blow to the mastoid region. Subsequent examination of the temporal bone revealed the middle ear, labyrinthine capsule, and perilymphatic space to be essentially normal. Corti's organ was severely degenerated and the tectorial membrane flattened throughout. The spiral ganglion and cochlear nerve were severely degenerated in all coils. Reissner's membrane was depressed in some areas, and adherent to the basilar membrane or absent in others. The sacculus was torn. The epithelium of the macula sacculi and macula utriculi was flattened.

Fischer[2] describes a case in which head injury and deafness occurred without fracture 15 years before death. There was complete destruction of Corti's organ, spiral ganglion and cochlear nerve. The endolymphatic space was filled with connective tissue, and the scala vestibuli and scala tympani contained connective tissue and bone.

Nassulphis[11] noted sagging of Reissner's membrane, displacement of the tectorial membrane and degeneration of the organ of Corti, cochlear ganglion and cochlear nerve in a number of cases. The following case is especially noteworthy. The patient suffered an occipital head blow one year prior to death. There was a high-tone deafness in one ear. Histopathological study of this ear showed severe degeneration of all neural elements in the basal coil. Reissner's membrane was collapsed so that the cochlear duct was hardly recognizable in the basal region. There were also three small chip fractures of the labyrinthine capsule.

Schlittler, Voss, and Wittmaack also have demonstrated isolated chip fractures of the labyrinthine capsule in histological preparations of human temporal bones.

SUMMARY

Diminished hearing acuity may follow a blow to the head. In some of these cases there is a fracture of the temporal bone. The fracture may be either longitudinal or transverse to the long axis of the petrous pyramid.

A longitudinal fracture of the temporal bone is characterized by bleeding from the ear, rupture of the tympanic membrane, and a combined conduction and nerve type deafness. The conduction deafness is caused by direct injury of the middle ear structures and is partially or completely reversible.

Following a transverse fracture there is severe vertigo with nystagmus to the opposite side, hematotympanum, complete deafness and total loss of vestibular function in that ear. The fracture line extends through the inner ear and degeneration of the membranous labyrinth follows.

Deafness may also occur in an ear in which there is no clinical or roentgenological evidence of fracture of the temporal bone. The hearing loss is of the nerve type and most severe for the high tones, particularly the 4096 frequency. In severe injury all frequencies may be involved. This type of deafness is frequently seen in the ear on the side opposite to a temporal bone fracture.

Brunner, Stenger, and Wittmaack have shown in animals subjected to head blows that hemorrhage often occurs into the perilymphatic spaces. Bleeding into the auditory nerve is less common. Wittmaack found degeneration of the hair cells and spiral ganglion to be most severe in the middle cochlear coil in cats subjected to head blows. He conjectured that a traveling pressure wave injured these structures directly.

A number of temporal bones have been described in the literature from patients having a deafness following head injury without fracture. Some have shown connective tissue and bone in the perilymphatic spaces with degeneration of Corti's organ and the spiral ganglion. Several have shown only severe degeneration of Corti's organ and the spiral ganglion.

Seventeen cases of deafness following head injury have been included in this report. The audiograms of many of these patients demonstrate a hearing loss which is most severe in the region of the 4096 frequency. These audiograms are similar to those of patients suffering from acoustic trauma due to air-borne sounds of high intensity.

A subsequent report will deal with an experimental study on cats conditioned to sound which have been subjected to head blows. The postconcussion audiogram of each animal will be correlated with the histological findings in the inner ear and brain.

UNIVERSITY OF CHICAGO.

TABLE 1.

DIFFERENTIAL DIAGNOSIS

	LONGITUDINAL FRACTURE	TRANSVERSE FRACTURE	INNER EAR CONCUSSION
Bleeding from the ear	very common	rare	never
Injury to the external auditory canal	occasionally	never	never
Rupture of the tympanic membrane	very common	rare; hematotympanum common	never
Cerebrospinal otorrhea	occasionally	occasionally	never
Facial nerve injury	25% of cases; usually temporary	50% of cases, often permanent	never
Hearing loss	combined conduction and nerve type deafness, all degrees; partial recovery	profound nerve type deafness, no recovery	all degrees, partial to complete recovery
Vertigo	occasionally; mild transient attacks	severe; nystagmus to opposite side; subsides in 2-6 weeks	normal; occasionally mild transient attacks
Vestibular function	normal; occasionally mildly depressed	no response	normal; occasionally mild depression
Roentgenological findings	fracture line frequently seen in squamosa or mastoid cells	fracture line through pyramid seen in 50% of cases	negative

REFERENCES

1. Alexander, A. F., and Scholl, R.: Beschwerden und Störungen in Hör— und Gleichgewichtsorgan bei der Nachuntersuchung Schädelverletzter, Monatschr. f. Ohrenh. 72:1021, 1938.

2. Fischer, J., and Wolfsen, L. E.: The Inner Ear, New York, Grune and Stratton, 1943.

3. Rutin, E.: Zur Klinik der Schlafenbeinbruche, Monatschr. f. Ohrenh. 71:179, 1937.

4. Grove, W. E.: Skull Fractures Involving the Ear. A Clinical Study of 211 Cases, Laryngoscope 49:678-707 (Aug.); 833-867 (Sept.) 1939.

5. Ulrich, K.: Verletzungen des Gehörsorgans bei Schädelbasisfrakturen, Acta Oto-laryng., Supp. VI, 1926.

6. Escher, Franz: Die Otologische Beurteilung des Schädeltraumatikers, Pract. Oto-rhino-laryng. 10: Suppl. 1, 1948.

7. Brunner, H.: Disturbances of the Function of the Ear after Concussion of the Brain, Laryngoscope 50:921, 1940.

8. Sakai, K.: Anatomische Befunde am Menschlichen Gehörorgan nach Basisfracture, Arch. f. Ohrenh. 85:188, 1911.

9. Barnick, Otto: Uber Brüche des Schädelgrundes und die durch sie bedingten Blutungen in das Ohrlabyrinth, Arch. f. Ohrenh. 43:23, 1897.

10. Kelemen, George: Fractures of the Temporal Bone, Arch. Otolaryng. 40:333-337 (Nov.) 1944.

11. Nassulphis, P.: Die Schädigung des Innenohres u. seiner Nerven nach Schädeltrauma, Mschr. Ohr.-Heilk. Jahrg. 79-80:68-86 (Feb.); 222-252 (June) 1946.

12. Voss, O.: Die Chirurgie der Schädelbasisfracturen, Leipzig, Ambrosius Barth, 1936.

13. Ecker, A.: Cerebrospinal Rhinorrhea by Way of the Eustachian Tube, J. Neurosurg. 4:177-178, 1947.

14. Klingenberg, A.: Die Isolierte Schneckenfractur bei Schädelbasisbrüchen, Ztsch. f. Hals-, Nasen-, u. Ohrenh. 22:452, 1929.

15. DeKleyn, A., and Stenvers: Uber die Technik der Rontgenologie der Augenhöhle und des Felsenbeines, Arch. Ohrenh. 103:162, 1919.

16. Nager, F.: Zur Histologie der Isolierten Schneckenfractur, Pract. Otorhino-laryng. 11:134-141, 1949.

17. Hofmann, L.: Fracturen der Schläfenbein pyramide, Zbl. Ohrenh. 7:539, 1925.

18. Schlittler, E.: Labyrinthzersplitterung-isolierte Vestibularfractur — Spätmeningitis nach 16 Jahren, Acta Oto-laryng. 24:213-221, 1936.

19. Stenger, P.: Beitrag zur Kenntnis der nach Kopfverletzungen auftretenden Veränderungen im inneren Ohr, Arch. f. Ohrenh. 79:43, 1909.

20. Klestadt, A.: Spätmeningitis nach Labyrinthfractur, Ztsch. f. Ohrenh. 69:229, 1913.

21. Mannasse, P.: Schädelbasisfracture, Beitr. z. Anat. Physiol. Path. u. Therap. d. Ohres. 21:230, 1924.

22. Perlman, H. B.: Process in Healing in Injuries to Capsule of Labyrinth, Arch. Otolaryng. 29:287-305, 1939.

23. Mayer, O.: Basalfissur, Monatschr. f. Ohrenh. 44:238, 1910.

24. Bast, T. H.: Development of the Otic Capsule, Arch. Otolaryng. 33:4, 1940.

25. Nager, F. R.: Beitrage zur Histologie der Erworbenen Taubstummheit, Ztschr. f. Ohrenh. 54:217-244, 1907.

26. Politzer, A.: Menierescher Symptomencomplex infolge traumatischer Labyrinthläsion, Arch. f. Ohrenh. 41:165-175, 1896; Lehrbuch der Ohrenheilkunde, p. 647.

27. Passow, A.: Die Verletzungen des Ohres, Wiesbaden, Bermann, 1905.

28. Schwartze, H.: Handb. d. Ohrenh., Leipzig, F. C. W. Vogel, 1892.

29. Schonbauer, L., and Brunner, H.: Schädelbasisbrüche, Handb. d. Neurol. d. Ohres 2:327, 1928.

30. Brunner, Hans: Erkrankungen des Innenorhes nach Stumpfen Schädeltraumen, Monatschr. f. Ohrenh. 59:697, 763, 922, 1925.

31. Wittmaack, K.: Uber die Traumatische Labyrinthdegeneration, Arch. f. Ohren-, Nasen-, u. Kehlkopf. 131:59-124, 1932.

32. Theodore, E.: Beitrag zur Pathologie der Labyrinthershütterung, Ztsch. f. Ohrenh. 61:299, 1910.

33. Uffenorde, W.: Histologishe Befunde, Beitr. z. Anat., Physiol., Path. u. therap. d. Ohres. 21:282, 1924.

34. Koch, J.: Studien Uber Veranderungen des Gehörorgans, inbesondere Störungen der Innenohrfunction nach Schädelunfallen mit und ohne Verletzung des Schläfenbeins, Arch. f. Ohrenh. 137:105-160, 1933.

35. Ruedi, L., and Furrer, W.: Das akustische trauma, Pract. Oto-rhino-laryng., 8: Fasc. 4, Suppl., 1946.

36. Linck, A.: Beitrag zur klinik und Pathologie der Schädelbasisfrakturen, Ztschr. f. Ohrenh. 81:265-306, 1921.

37. Oppenheim, H.: Lehrbuch d. Nervenkrank, Berlin, S. Karger, p. 1001, 1902.

38. Tobey, H. G.: Symposium, Neural Mechanism of Hearing, Laryngoscope 47:598-602 (Aug.) 1937.

39. Alexander, G.: Die Schussvertletzungen des Ohres, Handb. d. Neurol. d. Ohres. 2:449, 1928.

40. Hellmann, K.: Zur Pathologishen Anatomie der Taubheit nach Kopfschuss, Ztsch. f. Hals-, Nasen- u. Ohrenh. 1:358-370, 1922.

XXX

THE INCIDENCE OF TOTAL LARYNGECTOMY

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As part of a recent study dealing with the problem of speech rehabilitation in laryngectomized patients, a survey was made in an effort to determine the frequency with which the operation of total laryngectomy is being performed. The study was begun in 1948 and, therefore, the year 1947 was selected as the base period for this investigation. At first it was intended to limit the survey to the United States, but as the work progressed, a few inquiries were sent to certain foreign countries and interesting data obtained. The figures for the United States are believed to be reasonably complete for the year 1947. The reports from foreign countries are somewhat fragmentary. The figures herein reported could be used for comparison with similar surveys of the future to demonstrate changing trends (if any) in the management of laryngeal cancer.

Method of Conducting the Survey. Soon after the beginning of this investigation it was plain that the majority of total laryngectomies are being performed in the larger hospitals in the principal cities and on clinical services especially equipped to undertake this procedure. Nevertheless, in order to make the study complete, all cities in the United States with a population of 50,000 or more were polled.

At least one letter was written to a surgeon in each of these cities, selected whenever possible from those known to be interested in laryngeal cancer. Directors of tumor clinics and members of the American Board of Otolaryngology of the respective localities were also solicited for information. As a test of accuracy, independent inquiries were often sent to two or more individuals in the same city. The figures in the latter instances were usually identical. In general, the responses were prompt and satisfactory. Occasionally, additional information was supplied listing the specific hospitals in a given city with the number of operations in each. In other instances the figures for 1948 were furnished as well as for 1947. In many of the larger cities, up to 250,000 in population, it was clearly stated that no laryngectomies had been performed during 1947. All

From the Head and Neck Service, Memorial Hospital, New York, N. Y.

TABLE 1.
INCIDENCE OF LARYNGECTOMY IN THE UNITED STATES FOR THE YEAR 1947—CITIES

New York	125	Memphis	5
Philadelphia	96	Nashville	5
Chicago	69	Oklahoma City	5
Rochester, Minn.	65	San Francisco	5
Pittsburgh	47	Denver	4
Cleveland	42	Indianapolis	4
Detroit	28	Omaha	4
New Orleans	27	Rochester, N. Y.	4
Boston	26	Providence	4
Baltimore	25	Houston	4
Newark	25	Birmingham	3
Iowa City	18	Seattle	3
Los Angeles	18	Winston-Salem, N. C.	3
Durham, N. C.	12	Albany	2
Washington, D. C.	12	Buffalo	2
Atlanta	11	Elizabeth, N. J.	2
Kansas City, Mo.	11	Jersey City	2
St. Louis	11	Syracuse	2
Cincinnati	9	Madison, Wis.	2
Richmond	9	Portland, Maine	2
St. Paul	9	Spokane	1
Galveston	8	Dallas	1
Minneapolis	8	Salt Lake City	1
Louisville	7	Charleston, S. C.	1
San Antonio	7	Chattanooga	1
Charlotte, N. C.	6	Glendale, Calif.	1
Charlottesville, Va.	6	Long Beach, Calif.	1
Columbus	6	Hartford	1
Lexington	6	Waterbury, Conn.	1
Little Rock	6	Miami	1
New Haven	6	Patterson, N. J.	1
Portland, Ore.	6	Vicksburg, Miss.	1
		Total	846

TABLE 2.

INCIDENCE OF LARYNGECTOMY IN THE UNITED STATES FOR THE YEAR 1947—STATES

Pennsylvania	143	Georgia	11
New York	135	Tennessee	11
Minnesota	82	Connecticut	8
Illinois	69	Arkansas	6
Ohio	57	Oregon	6
New Jersey	30	Oklahoma	5
Michigan	28	Colorado	4
Louisiana	27	Indiana	4
Massachusetts	26	Nebraska	4
California	25	Rhode Island	4
Maryland	25	Washington	4
Missouri	22	Alabama	3
North Carolina	21	Maine	2
Texas	20	Wisconsin	2
Iowa	18	Florida	1
Virginia	15	South Carolina	1
Kentucky	13	Utah	1
District of Columbia	12	Mississippi	1
		Total	846

negative data have been omitted from the tables; only those cities are listed from which positive information was obtained.

A few tentative inquiries to foreign countries resulted in such interesting data that further efforts were made to poll certain European countries, or at least the larger cities. In the cases of Sweden and Denmark the statistics are probably complete for the country as a whole. The totals for London and Paris were obtained from data given in considerable detail from reliable sources and are believed to be reasonably correct.

Interpretation of Data. As seen in Table 1 the operation of total laryngectomy was performed in at least 846 cases in the United States during the year 1947. Obviously, additional single instances of this operation may have been performed in smaller hospitals and

TABLE 3.

INCIDENCE OF LARYNGECTOMY IN FOREIGN COUNTRIES FOR THE YEAR 1947

(INCOMPLETE CENSUS)

ARGENTINA		91
Buenos Aires	71	
Córdoba	6	
Rosario	14	
AUSTRALIA—(Sydney)		2
CANADA		7
Halifax	2	
Montreal	5	
CZECHOSLOVAKIA—(Prague)		25
CHILE—(Santiago)		3
CUBA—(Havana)		12
DENMARK—(Copenhagen)		11
GREAT BRITAIN		35
Edinburgh	5	
Liverpool	5	
Birmingham	7	
London	18	
FRANCE		115
Paris	95	
Montpellier	20	
HAWAII*—(Honolulu)		2
INDIA—(Bombay)		6
ITALY		28
Florence	4	
Padova	24	
PUERTO RICO*		3
PORTUGAL		2
Coimbra	1	
Lisbon	1	
SPAIN—(Madrid)		122
SWEDEN—(Stockholm)		20
URUGUAY—(Montevideo)		31
VENEZUELA—(Caracas)		13
TOTAL		528

*U. S. Possession

in smaller cities, a complete record of which would be difficult to obtain by a survey. So far as the figures herein reported are concerned, it is felt that the comparative geographic incidence as cited in this study is undoubtedly correct, even though the total number of operations as given may be slightly less than the actual number.

The statistics in the accompanying tables represent positive information. The numbers of operations performed in the respective cities (Table 1) and states (Table 2) are presented in order of their relative frequency. Reference to these data indicates that total laryngectomy is most frequently performed in the larger cities of the East and Midwest, particularly in the larger clinics. The more specific data (not detailed in this report) indicate that the operation is performed in highly organized clinics and is seldom performed outside hospitals not specially equipped for this purpose. Apparently, the higher incidence of the operation in certain cities is due largely to the activities of a single clinic, sometimes even to the activities of a single surgeon.

As regards the foreign countries, it is of interest to note the relative infrequency with which the operation is performed in London as compared with Paris and Madrid. It is also of interest to note the relatively large incidence of laryngectomy, with respect to the population, in such cities as Montevideo, Buenos Aires, and Caracas (Table 3).

SUMMARY AND CONCLUSIONS

The operation of total laryngectomy was performed in approximately 846 instances in the United States during the year 1947. A greater number of such operations were performed in the larger cities, particularly in the larger clinics. Some sections of the country are sparsely populated or contain few large cities; in these areas proper facilities for the operation may not be available, and, therefore, laryngectomy is seldom performed in such localities. Whenever figures for 1948 were supplied, the number of operations for 1948 was larger than that reported for 1947. This finding might indicate that the use of laryngectomy in the treatment of cancer is on the increase. A comparison of the present figures with a similar survey in five or ten years would be of considerable interest.

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XXXI

THE OFFICE TREATMENT OF CHRONIC OTITIS

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It is to be appreciated that the results of the office treatment of chronic ear infections does not approximate, in many instances, the spectacular results that surgery of the ear obtains in clearing up the chronically discharging middle ear.

It is our constant endeavor with our present knowledge and the application of the newer antibiotics in otologic treatment to prevent a radical procedure if at all possible. Research investigation of diseases of the ear is difficult; however, the controlled studies of Senturia[1, 2] and others,[3-6] with this goal in view is a move in the right direction—they are to be congratulated.

Our treatment is aided here, as elsewhere in the body, by a clear understanding of the embryological and histological origin of the areas affected. The ectodermal covering of the first visceral cleft later forms a continuous epithelial covering over the external auditory canal and tympanic membrane. The outer part of this lining is protected by hairs and their follicles into which open the modified sweat glands secreting cerumen. The inner part of the lining is thinner, intimately adherent to the periosteum, and is devoid of these glands except on the posterosuperior surface so it is not protected to the same degree from trauma. The function of this lining is to receive sensory impulses from the outside, to excrete certain substances, to keep the cavity warm and to protect the hearing mechanism.

The outer layer of the epidermis called the stratum corneum or horny layer contains keratin and is resistant to infection maintaining a dry surface over the tympanic membrane and adjacent canal wall.

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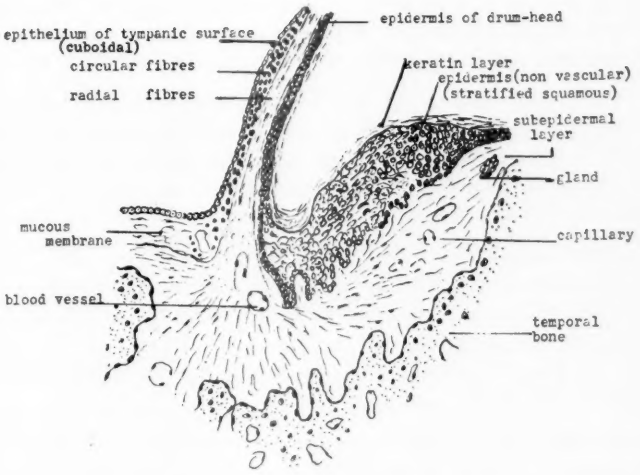

Fig. 1.—A drawing of a low power photomicrograph of a section including the external ear canal, the tympanic membrane, the middle ear mucosa and the bone surrounding these structures.

It extends down as far as the opening of a sebaceous gland. The ceruminous glands, however, extend down to the perichondrium and consist of secretory and excretory portions. The deeper layers seen in skin elsewhere are lacking in this region. The epidermis of the inner one-third of the canal has been measured at less than 0.07 mm in thickness and it is possible that traumatic dry wiping could easily damage this area[7] (Fig. 1). The Indians learned that when wax removed from the ear was applied immediately to a wasp sting that the pain and swelling was relieved. The nerve supply arises chiefly from the mandibular branch of the trigeminal and from the sensory portion of the vagus nerves with terminal branches in the epidermis. Pain arises from pressure or baring of the naked nerve endings lying in the deeper layers of the epidermis. The variation of a pleasurable response from tickling of the ear as used in the quaint Oriental custom—to the symptom of itching with or without visible pathology—to shock from the extreme pain of local inflammation—is evidence of the adequate nerve supply. An edematous external canal arises from lymphatic and venous compression by a lodged foreign body or inflammatory response. Temperature readings of the inner one-third of the ear canal in ten normal subjects averaged 98.2° F. and there was a difference of as much as 0.8° F. in the two ears of one subject at normal room temperature.

With the use of a universal indicator (B. D. H.) we found that the pH of the skin of the normal ear canal falls on the acid side in the region of 6.4 and may extend from a pH of 5 to 7.8.

With a high pH in an otherwise normal ear canal one must eliminate the possible presence of soap remaining from the morning toilette. Marchionini[8] demonstrated that vesicular eczema with or without resultant fissure formation produces an alkaline pH and he differentiates between a so-called normal "acid mantle" and an "alkaline mantle" as seen in seborrheic conditions.

The conditions which interfere with normal skin function and its defense mechanisms are chiefly due to a change in temperature or humidity, the introduction of a foreign substance or the entry of bacteria into the subepithelial layer with the formation of their destructive proteolytic enzymes.

The effect of trauma cannot be overemphasized. The average person scratches his ear once or twice a day and is usually totally unconscious of doing so, and in addition many make it a habit to clean their ears with any available object. This is the quickest way to break the surface continuity of the protective layer of the skin and allow the entry of bacteria. Bacteria generally grow best at a temperature of 68° F. and with a rise in humidity and temperature there is a rise in nature's bacterial floral growth.

Covering the ears appears to raise the relative humidity of the region and from the general population the number of nuns with ear complaints is amazing. Veterinarians tell us that spaniels suffer greatly from severe ear infections—one wonders if there is also a decrease in the blood supply due to the bending over of the ear in these dogs.

When accumulated or impacted cerumen comes in contact with water it swells causing an interference with blood supply and venous return, also it retains moisture locally in this ideal and relatively anaerobic and dark test tube. Self-inflicted trauma in removal of this water after swimming may also play a part. The introduction of clean water when syringing out accumulated cerumen is often followed by infection when careless technique is used; presumably a break in the skin occurs allowing the entry of bacteria.

The history of the poliomyelitis victim may be associated with a change in body temperature after swimming or chilling—this history can be elicited occasionally in cases of external otitis.

Dietary changes aid staphylococcal infections to gain a foothold and poor living conditions are often associated with aural furuncu-

losis. One of the earliest manifestations of vitamin A deficiency is dryness of the skin; the sebaceous glands and sweat glands atrophy with suppression of their secretions followed by an eruption of the skin due to changes in the hair follicles.[9]

Carelessness in drying the ears after washing or shaving leaves a layer of soap which interferes with the acid mantle and continuation of this practice cannot but damage the skin at the external auditory meatus.

Certain individuals suffer from sensitiveness to cosmetics, contact of clothing and food allergy. A severe allergic reaction causes an upheaval in the local physiology.

It is possible to find on culture of material from the ear canal any or all of the normal flora of the skin. However, in the normal ear most investigators have demonstrated the more or less constant appearance of *Staphylococcus aureus* and *Staphylococcus albus* and the diphtheroids, but only in disease do *Pseudomonas aeruginosa* and various fungi appear.[10-12] The latter are nearly always present in the air but require the necessary pH and living conditions for their saprophytic growth and are occasionally cultured from cases of external otitis.

An existent or acquired allergy to certain bacterial toxins and fungi produce the so-called id reaction. It would be of interest to know how many patients with active fungus growth in the ear also suffer or suffered from trichophyton. Many dermatophytoses pass unnoted with the passing symptom of mild pruritus.

To summarize, the skin of the external canal is potentially subject to any pathological change occurring elsewhere in the body. Whatever the cause, the loss of keratin, the entry of bacteria, the production of bacterial toxin with proteolysis and maceration then leaves the remaining forces dependent on body resistance and the maintenance of an adequate blood supply.

A simple classification of external otitis is as follows:

1. Furunculosis
2. Otitis externa diffusa
3. Eczematous and allergic otitis—hyperkeratotic or exudative
4. Otomycoses

1. *Furunculosis.* Furunculosis is an abscess of a hair follicle caused by the entry of *Staphylococcus aureus* at the point where a sebaceous gland duct meets the protective keratin layer; its entry

is facilitated by repeated local friction. The lethal toxin destroys the adjacent capillaries with the release of fibrin producing local tension and intense pain. In addition the patient complains of a fullness in the ear and a slight hearing loss if the edema is marked. Removal of the superficial necrotic debris is carried out by gentle irrigation or suction and this is an almost painless procedure. Incision or local injection is avoided. The skin is dried with 95% alcohol and a thin coating of tincture zephiran 1:1000 which is dried from the skin with air. This leaves a superficial surgically clean area preventing adjacent follicle infection. If there is edema a small cotton wick of aluminum acetate 1% is gently inserted with a larger piece similarly soaked at the conchal orifice. The bacteriocidal and hygroscopic action is maintained by having the patient keep the wick saturated for 24 hours with this solution. Aluminum acetate does damage the superficial skin to a degree but is less irritating than glycerin compounds, and if a repeat procedure of sterilization and drying is carried out after removal of the wick this is reduced to a minimum. Heat may be applied locally, penicillin intramuscularly, with codeine and aspirin given for pain, to control the condition until the natural responses take over. The fractional use of insulin may be recommended in recurrent furunculosis. The resistance of man to this ubiquitous parasite is evidently high or it would long ago have destroyed our race. A spreading surface cellulitis from streptococcal infections responds rapidly to penicillin.

2. *Otitis Externa Diffusa.* The presence of moisture and the breakdown of the protecting keratin layer, which consists of an albuminoid type of protein, produces maceration with a change in the pH to the alkaline side. Thus ideal conditions for a spreading infection exist. The presence of bacteria in this ideal culture medium rapidly overwhelms nature's barriers, producing a diffuse surface infection. Cultures from the ear reveal gram-positive organisms, principally *Staphylococcus aureus* and *Staphylococcus albus* with the diphtheroids and gram-negative organisms, chiefly *Pseudomonas aeruginosa,* and in some cases various fungi. Cultures should be taken if bacteriological facilities present themselves but in the average practice it is not practical; however, in cases which do not respond rapidly to treatment the offending organisms must be identified since further therapy cannot be properly directed.

The less damage done to this already sensitized area the better, and unfortunately the skin of the ear is a small area where the physician and patient always overtreat the disease producing a prolongation of the infection. In diffuse infections the pH of the area is altered with the formation of an "alkaline mantle" from proteo-

lytic hydrolysis, and in addition suppuration produces a local damaging histamine reaction in the surrounding tissues. To treat this type of ear the dead or dying tissue with its bacterial products must be removed and the moist conditions necessary for growth altered. Unfortunately the common gram-negative pseudomonas is very resistant to the known antibiotics. It is highly motile in culture and is a saprophyte of water, hence the value of syringing out the ear with saline since the organism is soluble in water. It is resistant to cold and will multiply at 32° F., so it is called psychrophilic, or cold-loving, in contrast to most bacteria whose optimum growth occurs at 68° F., approximating room temperature.[13] The odor of *B. pyocyaneus* in growth is characteristic; each physician should visit the bacteriology department and ask to smell a culture, it is so typical.

The treatment is carried out by gentle suction or irrigation with 5% saline. It has been found that 10% saline inhibits the growth of saprophytes but since this concentration is irritating, saline in weaker concentration is used as a feeble antiseptic which dehydrates the proteins of the skin, producing poor culture material and also dissolving certain bacteria. The area is then dried with further suction and the application of 95% alcohol. A thin layer of aqueous gentian violet and 95% alcohol is applied and the skin dried with a stream of air. After five minutes an exudation of fluid may occur from the irritating effect of the gentian violet and this must be removed. Treatment every two days for a week generally clears the condition. Spores are resistant to drying and since the life cycle is estimated at two weeks, vigilance is the watchword. The patient should make weekly visits for a period of a month. This dry treatment restores the pH to the acid side of normal and it has been found that bacteria grow poorly at a pH of 6 and cease to grow at a pH below 5 and above 9.

Sulfanilamide powder may be frosted over the surface by powder insufflation after the above drying treatment and gratifying results occur if it is used in the early treatment only. Treatment carried out by the patient at home is generally unsatisfactory; however, if he lives out of town, 95% alcohol drops or tincture zephiran 1:1000 are used for one week, twice daily. There are no hard and fast rules but cleansing of the cavity and avoidance of further unnecessary trauma are the main observance. This must be absolutely clear to the patient as well.

3. *Eczematous and Allergic Otitis.* In the eczematous type of infection there is an exfoliation and tendency to crusting, later an exudation with fissure formation. The alkaline pH produced favors the growth of bacteria, so an early eczema caused by a sebor-

rheic dermatitis or allergic manifestation may progress easily into a pyogenic infection. When the patient is treated and the purulent condition cleared the allergy may remain and the physician wonders why the ear does not dry up. Treatment of eczema responds quickly to gentian violet 1% aqueous and 95% alcohol. This method of application insures the gentian violet is fresh since alcoholic solutions evaporate and produce an irritating concentration of the dye. If the pinna is affected, ammoniated mercury 2% heals the fissures and removes the hyperkeratotic areas. This ointment should be removed daily with household vinegar before a fresh application is made, thereby constantly changing the pH. To restore the horny layer to normal, exaurol, pragmatar or salicylic acid 2% in 70% alcohol relieves the pruritic aftermath. Sulfur compounds restore the cystine content to the keratin layer by local application. If tincture zephiran is applied to the affected surface prior to the ointment application it leaves a sterile area over the epithelium and neutralizes the alkaline ointment to a degree.

The causative substances producing an allergic manifestation when investigated and eliminated give prompt relief. Many of our local treatments when used over a period produce a local sensitivity and should be stopped short of this occurrence instead of continuing them with renewed vigor. We have observed so many local reactions to penicillin, sulfa compounds, streptomycin and other substances such as Dibromsalicylaldehyde that we use them with great caution and reserve their use for cases that do not clear with the treatment previously described. If iodine powder is used, mercurials should be avoided as a severe reaction of the skin occurs when these drugs meet. Substances such as orris root, perfumes, fur, wool, irritating soaps are common offenders. Unless these are eliminated the return of secondary infection will occur.

4. *Otomycosis.* Otomycosis is defined as an exudative inflammation and pruritus of the external auditory meatus from which can be isolated a varying number of fungi. Several investigators have elucidated the fact that the number of fungus infections occurring in the ear are greatly overestimated. Fungi, however, are everywhere and moisture is a necessary element but their spores are quite resistant to drying. In the syndrome of otomycosis it is difficult to evaluate the significance of the various fungi which have been isolated; those most commonly seen are aspergillus, actinomyces, penicillium and mucor. It is of interest that these fungi cannot be cultured from normal skin and rarely, if ever, can be found in the normal ear canal. They are actually of secondary importance in consideration of the causation of infection. Too little attention is paid

to the coexistence of *Staphylococcus aureus* and *B. pyocyaneus;* and the fungi, contributing to the maintenance of such organisms. We have found the most common invaders in our locality to be aspergillus, *Candida albicans* and occasionally monilia.[14] The portal of entry of fungi is through the air, with dust and hairs, epidermal scales and water. Living tissue is not as a rule primarily invaded but is greatly irritated by their presence. The inflammation is accompanied by exfoliation of the superficial epithelium with the formation of epithelial debris and mycelial strands, and the associated bacterial invasion of the skin causes suppuration and food for fungus growth.

An easy diagnostic procedure for office use is to dissolve some epithelial debris in 10% potassium hydroxide by heating, after which the characteristics of the mycelium and spores can be seen under the microscope. Cultures can be taken if this is unsuccessful and the species identified. The differential diagnosis of cellulitis, seborrheic dermatitis, impetigo and contact dermatitis must be considered. Treatment in this condition requires removal of all cerumen and debris by gentle irrigation and drying followed by the application of tincture zephiran 1:1000 to remove the pyogenic organisms. This solution has a pH of 6.5-6.7 and is followed by a wick soaked in aluminum acetate 1% which has a pH of 4-4.5. This is left in contact with the area for 24 hours and is kept moist with this solution. In this way there is maintained a constant fungicidal action together with softening of the surface debris. After removal, the skin is dried with 95% alcohol and gentian violet 1% aqueous is applied and the canal again dried. Daily treatments for four days generally produce gratifying results, then the case is followed carefully for the growth of any remaining spores. The patient is warned to guard against the possible entry of water into the ear. If gentian violet is not producing results a shift to sulfanilamide powder for a few days or frosting of the skin with Gantrisin powder daily is of benefit in removing associated gram-negative organisms.

We do not claim better results than other physicians but our results are consistent in their response to the *modified dry treatment.* Any application usually results in a change of the pH which temporarily halts the bacterial or fungus growth, hence the literature abounds in substances, all of which may be initially beneficial.[1, 4, 10] A comparison with the history of the treatment of burns is of great interest when one notes the phases through which it has passed. Minimal débridement with the avoidance of further trauma, gentle cleansing of the surface and leaving the rest to nature is now the

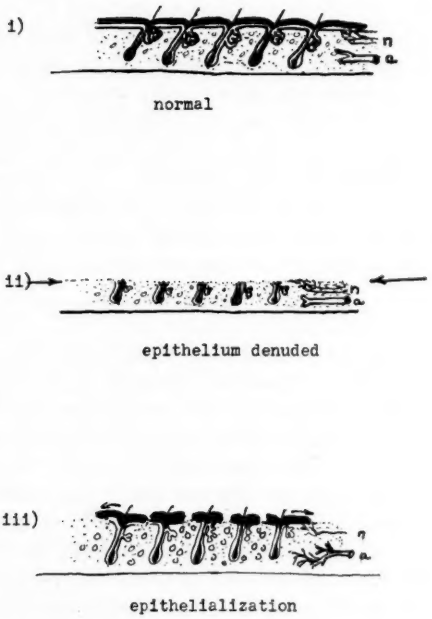

Fig. 2.—This diagram illustrates the source of regeneration of epithelium from around the hair follicles and duct epithelium following tissue disintegration following infection.

accepted treatment. One notes the avoidance of the antibiotic substances in local treatment (Fig. 2).

To Recapitulate:

1. The treatment of infections of the external ear depends on the existing condition. Interference with nature's reparative processes meets with disaster and one should not add insult to injury by intensive local applications.

2. The removal of the epithelial debris and enzymal surface products by gentle irrigation or packs saturated with mildly hypertonic saline followed by drying hastens the return of a normal acid pH and the subsequent death of the saprophytes.

3. The follow-up routine is a necessity since recurrence is frequent when one is dealing with the gram-negative organisms. These organisms are the most important invaders in external otitis as we see it today.

4. The health of the regenerating cells reflect the health of the patient and advice in this direction is almost as important as local treatment.

5. The importance of diagnosing allergic sensitivity when it occurs cannot be overemphasized.

6. The co-operation of the patient is absolutely essential.

The treatment of the chronically discharging middle ear depends on an understanding of the histological and physiological problems concerned.

The lining of the eustachian tube, middle ear and mastoid cells arises from the entodermal invagination of the tubotympanic recess. The epithelial covering varies from ciliated columnar in the auditory tube to cuboidal or columnar in the middle ear cleft. The area in the middle ear close to the auditory tube is also ciliated and the middle ear bones are surrounded by simple squamous epithelium.

The reaction to infection in this region is similar to that evoked elsewhere in the body. A simple review of the inflammatory process is necessary to explain the principles of treatment of these infections with the antibiotics and other items in an office armamentarium.

Organisms enter the middle ear cavity via the eustachian tube and produce an inflammation of the tube and middle ear. There are few pauses and much overlapping in the process of injury with the subsequent response of the tissue to the repair process. The toxic by-products produce a destruction of the mucous membrane with an increase in capillary proliferation in the subepithelial tissues. There is a leukocytic margination along the wall of the vessel and the exudation of fluid through the walls of the capillaries produces an edema of the tissues from the escape of blood. Fibrin forms, and young undifferentiated mesenchymal cells grow out along these fibrous bands producing organization of the tissue as a defense mechanism. The proliferation of young capillaries attempting to carry off the waste products cannot grow downwards into the bony wall and therefore grows outward, producing the familiar humped up areas of granulation tissue (Fig. 3). It is difficult for re-epithelialization to occur since there are no hair follicles or ducts and new skin must grow in from the margins of the denuded areas. Granulation areas indicate the point of greatest local suppuration and present a powerful barrier to septicemia. This effort may out-run itself in the formation of polypoid material and subsequent degeneration of the capillary and lymphatic elements. Rubbing the surface of a granulation causes rupture of the capillary loops, materially inter-

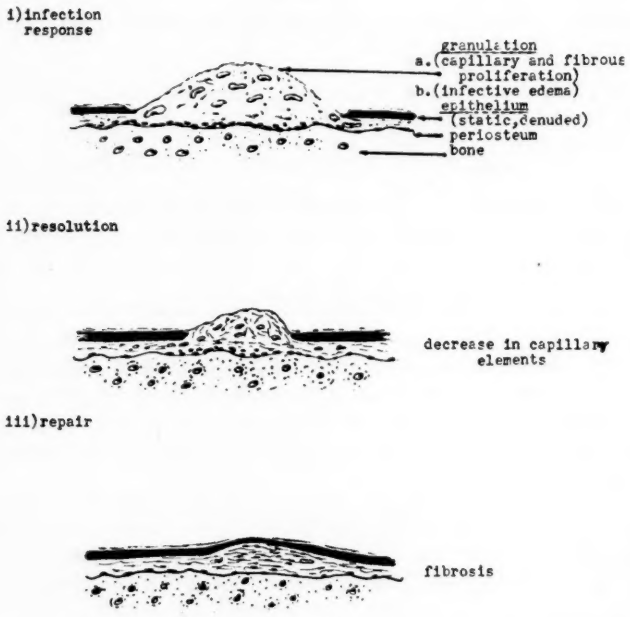

Fig. 3.—A diagram showing the response of the middle ear mucosa to infection and the subsequent slow peripheral epithelialization in the absence of hair follicle regenerating elements.

fering with the process of healing. Not until the leukocytic response has overcome the infection does the epithelium begin to cover the surface and healing can be said to have begun. In the superficial layers the fibroblasts grow at right angles to the surface while deeper they are parallel to the surface, so removal of the outer four-fifths or more of the granulation area promotes more rapid healing. When the wound is aseptic, epithelium will grow in from the edge in two or three days but when the surface is bathed in pus circumferential epithelialization is retarded. When complete epithelialization has occurred the new vessels gradually disappear and the pearly white appearance of an almost avascular healed lesion appears. The continuance of a granulating middle ear is indicative of the presence of a still active bacterial flora and the inactivity of adequate lymphatic drainage to carry away the waste material. In such conditions epithelium will not grow, and when healing is long delayed epithelial processes grow down towards the periosteum with the formation of a walled off area and the conditions favorable to cholesteatoma.

The middle ear structures are not suspended in space since each retains a remnant of its mesentery-like folds indicating the origin of the part.[15] Contact between these inflamed folds with denudation of the surface epithelium on the applied surfaces results in irreversible adhesions. The fibrous bands contract with scarring and a permanent adhesive process results with loss of function. With our knowledge we should be able to prevent this occurrence.

Chronic infection may arise in two ways, either (1) from a subsiding acute infection wherein treatment is simple, or (2) from an infection of low intensity failing to call forth an acute response but stimulating cellular proliferation.

Treatment of the acute phase as in acute otitis media, whether of traumatic or infective origin, is now generally agreed treated best by conservatism. The main object is to leave nature alone and stay out of the external ear canal, providing drainage by eustachian tube therapy. Penicillin intramuscularly is still our standby and may be continued with oral sulfonamide therapy if a high fever exists. However, instead of a well timed paracentesis the tendency today is to let penicillin and Auralgan do all the work with the all too common resultant traumatic perforation. It often occurs at inopportune moments and the confidence of the relatives and patient is lost. Paracentesis with antibiotic therapy, ephedrine by mouth, and belladonna are usually prompt in their action. This treatment is modified for use in recurrent otitic barotrauma but aspiration of the middle ear contents is withheld awaiting some response from tubal inflations. These inflations also prevent the adhesions seen so often following the treatment of an acute ear by physicians using penicillin alone with or without nasal medication.

Once a perforation occurs the normal bacterial flora of the external ear canal become a potential danger and if they gain a foothold the chronically discharging ear becomes acute. In these cases, as shown by many observers, the present day situation is one wherein the *Pseudomonas pyocyanea* is the chief offending organism tending to chronicity. The molds may also be present.

The first attention in chronic otitis should be toward the eustachian tube. Sloughing of the cilia and epithelial lining produces denuded areas resulting in the formation of granulation tissue and a suppurative salpingitis.[16]

Constant irritation from middle ear drainage complicates healing and a scarred and permanently blocked windows may be the end result. Sinus therapy should be instituted when there is an associated sinusitis; the removal of adenoid tissue from the vault in-

cluding the lateral bands, followed by discreet irradiation of the tubal area is a necessity. The emphasis is placed on adequate surgery under direct vision. Frequently in a co-operative patient granulation tissue can be visualized in the tube one-fourth inch from the orifice. The use of ephedrine, gr. ¼, and atropine, gr. 1/500 by mouth, aids in reducing postoperative edema and hastens drainage of the middle ear.

In sections of the middle ear one sees engorged blood vessels with the epithelial lining reduced often to a single layer of cuboidal cells with a few or no ciliated cells remaining. The subepithelial tissue is thickened and fibrous connective tissue has together with endothelial elements produced local masses of granulation and exudate in the middle ear cleft.

Treatment of granulation tissue approximate to the pharyngotympanic tube is perhaps best effected with ear drops containing ephedrine. With the affected ear upward and by filling the canal with these drops pressure on the tragus produces suction and allows the drops to gain entrance to the tube as the granulations shrink. The drops also provide local medication.

If cerumen is present, removal with 3% stock hydrogen peroxide must be followed by 95% alcohol, after which the medication of choice may be introduced. We have found tincture zephiran 1:1000 to be immediately bacteriocidal and almost non-irritating, having a pH of 6.5; this solution can be used by the patient at home. When the condition is resolving, a small dry pack soaked in tincture zephiran and applied approximate but not blocking the perforation maintains a barrier to prevent further irritation of the external auditory meatus. In our experience the main offender in continuing suppurative ear conditions is *B. pyocyaneus.* This organism can be effectively dealt with by using aluminum acetate 1% as drops for a limited period or acriflavine 1:1000.

Failing conservative treatment, the removal of granulation tissue must be sufficiently adequate on the first attempt so as not to stimulate further granulation formation. As stated previously wiping the surface of granulations produces bleeding due to opening of the capillary loops which stimulates growth of these elements. A sterile suction of the middle ear contents with instillation of medication and removal of excess solution avoiding trauma to the granulations, followed by thorough drying is preferable. The use of hydrogen peroxide to stop bleeding is superior to silver nitrate applications which produce a devitalized area inviting further bacterial invasion. Pseudomonas infections also respond to gentian violet 1% aqueous

and 95% alcohol used discreetly over the inflamed tissues. This does not rid the cavity of pseudomonas but protects the surface of regenerating tissue from toxic proteolysis. (Acetic acid 1% with equal parts of 5% saline provides an adequate surface cleanser.) Aqueous gentian violet may be given in the form of ear drops for three to four days, but when used for a longer period it is irritating.

With inadequate middle ear drainage an invasion of the mastoid cells occurs and the suppurative process causes a destruction of the epithelial lining with connective tissue infiltration and the production of granulation tissue. The osteoblastic activity of the periosteal lining of these cells with new bone formation is an interesting pathological development producing the periantral sclerosis seen in the x-ray films of a chronic suppurative mastoiditis. The presence of embryonic mesenchymal masses is occasionally seen at operation and these have a potential ability to become invaded and softened, and produce a chronic discharge. The proliferation of epithelium through tympanic membrane perforations with subsequent cholesteatoma formation into the attic with extension posteriorly into the aditus and antrum has been observed frequently. Removal of a portion of this cholesteatoma is possible using a small curet or suction; however, as long as the epithelial lining remains, new formations will occur. Irrigation is not too satisfactory and is to be deprecated, following observation of surgical emergencies over a number of years. With the destruction of surrounding bone by a cholesteatoma, the varying symptoms of fullness, tinnitus, vertigo, headache and facial paralysis occur—the only treatment is in the operating theatre. Conservatism has no place here.

A word regarding allergic manifestations in the middle ear: Allergy is a term often used to cover ignorance or give a touch of mystification to the process, yet many destructive lesions are in reality a hypersensitiveness of the tissue to bacterial proteins. One should remember that redness of the presenting tissue does not exclude allergy and this process also explains in part the active response of tissue to an acute exacerbation in a chronically discharging ear. The initial lesion in the body may be insignificant and far removed, carried by the blood stream and producing an id reaction in the ear.

A smear of the cellular debris may be stained with Hastings stain and a study of the cellular components will clarify the type of reaction with which one is dealing.

Treatment of allergic conditions consists in stopping a treatment and starting fresh with simple cleansing and drying of the part affected. Antihistaminics and ephedrine by mouth aid in the

recovery. One wonders how the wholesale use of these antihistamines will change the middle ear pathology as we know it today.

SUMMARY AND CONCLUSIONS

The main therapeutic treatment of chronic otitis is:

1. Cleansing of the cavity with removal of the accumulated toxic products. This is carried out with as little insult to the involved areas as possible.

2. The maintenance of a dry cavity for as long a period as possible after the office visit.

3. The use of sulfonamides, penicillin and streptomycin powders for the most part should be reserved for postoperative cavities and even then used to a minimum. Granulation tissue appears to spread with their continued use.

4. If the pH of the middle ear can be regulated to the acid side it will shorten the healing process. However, a low pH injures the leukocytes and nature's responses are damaged, producing an increase in suppuration. Our office studies of pH are carried out with a B.D.H. Universal Indicator and a quick, easy estimation of the pH is possible. Electrical estimations are cumbersome and are not an office procedure; furthermore many feel the results are not as accurate as the Universal Indicator method. A cytological smear showing a shift to mononuclear cells indicates a rise in H-ion concentration.

5. At present we are reviewing the war treatment of wounds in an attempt to follow their success and apply it to our field. We are using irrigations in postoperative cavities with such substances as Hygeol 1:16 or acetic acid 1% with saline 5% and with other solutions with an alkaline pH; in changing the pH back and forth the bacterial flora are also changed. This is again followed by thorough drying of the cavity. These procedures are not as practical in fenestrated ears where an active fistula is present and this is also true in some postoperative radical mastoid cavities. In both of these cavities the *B. pyocyaneus* organisms are dominant.

A chronically discharging ear which can be made dry with office treatment is indeed gratifying to the patient. In closing, a word of caution: A new growth should never be missed if biopsies are performed of areas of active bleeding or areas presenting any odd characteristics. To miss this diagnosis is an unforgivable mistake.

Treatment consists of the following:

1. The removal of dead or dying tissue with its contained bacterial flora is carried out as gently as possible, by 5% saline irrigation or surface contact for five minutes. Surface dry wiping is not conducive to healing.

2. The complete drying of the cavity with 95% alcohol.

3. Adequate removal of excess granulation tissue.

4. The application of a dye to produce a protein precipitate protecting the surface and allowing for an ingrowth of epithelium beneath the crust.

5. Encouragement of cellular activity by maintenance of an acid pH and an improvement in local circulation.

6. A minimum of trauma and avoidance of overtreatment.

7. The exclusion of antibiotics locally, if possible.

8. The cessation of all treatment (except 1 and 2 above) if flare-ups continue with medication.

9. A careful study of associated infected foci of infection.

With careful management the prevention of adhesive processes from this time is a possibility.

The general health of the patient is most important.

Each case is an individual problem.

A study of pathological processes in the ear and their treatment has been presented.

174 St. George St.

REFERENCES

1. Senturia, Ben H.: Treatment of External Otitis; I. Local Sulfonamide Therapy, Laryngoscope 54:277, 1944.

2. Senturia, B. H.: Etiology of External Otitis, Laryngoscope 55:277 (June) 1945.

3. Fabricant, N. D., and Perlstein, M. A.: PH of the Cutaneous Surface of the External Auditory Canal, Arch. Otolaryng. 49:201 (Feb.) 1949.

4. Syverton, J. T., Hess, W. R., and Krafchuk, J.: Otitis Externa: Clinical Observations and Microbiologic Flora, Arch. Otolaryng. 43:213 (Mar.) 1946.

5. Fischer, J., Department of Health Provincial Laboratories, Toronto: Personal communication, 1947-1950.

6. Hayes, M. B., and Hall, C. F.: The Management of Otogenic Infection, Tr. Am. Acad. Ophth. and Otolaryng. Jan-Feb., 1947, pp. 149-163.

7. Ham, Arthur W.: Textbook of Histology, Philadelphia, J. B. Lippincott & Co., 1950.

8. Marchionini, A., and Hausknecht, W.: Sauermentel der Haut und Bakterienabwehr; I. Die regionare Verschiedenheit der Wassersstoffionenkonzentration der Hautoberflache, Klin. Wchnschr. 17:663 (May 7) 1938; II. Uber die regionare Verschiedenheit der Bakterienabwehr und Desinfektionskraft der Hautoberflache, 736 (May 21) 1938.

9. Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice, Ed. 3, Baltimore, The Williams and Wilkins Co., 1950.

10. Fowler, E. P., Jr.: Topical Applications to the Skin of the Ear Canal, Tr. Am. Acad. Ophth. and Otolaryng. July-Aug., 1949, pp. 637-641.

11. Nelson, R. F.: External Otitis in the South Pacific, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 54:367 (June) 1945.

12. Salvin, S. B., and Lewis, M. L.: External Otitis with Additional Studies on the Genus Pseudomonas, J. Bacteriol. 51:495 (Apr.) 1946.

13. Gardner, A. D.: Bacteriology, London, Oxford University Press, 1938.

14. Hudson, Arthur, Toronto: Personal communication.

15. Grant, J. C. B.: Method of Anatomy, Ed. 4, Baltimore, The Williams and Wilkins Co., 1947.

16. Eggston, A. A., and Wolff, A. B.: Histopathology of the Ear, Nose and Throat, Baltimore, The Williams and Wilkins Co., 1947.

XXXII

THYROGLOSSAL DUCT CYSTS

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A study was made of 128 cases of thyroglossal duct cysts which were admitted to the Massachusetts Eye and Ear Infirmary and to the Massachusetts General Hospital from 1937 to 1949. To better understand the clinical aspects, a brief review of the embryology is included.

Embryology. Unlike the branchial cleft cysts (lateral cervical cysts), entities whose origins are still under debate, the development of thyroglossal duct cysts is generally accepted.[1-6] During the fourth fetal week there is an evagination of the floor of the primitive pharynx in the midline, this evagination representing the anlage of the isthmus and part of the lateral lobe of the thyroid gland.[7, 8] Growth continues down, forming in its progress a duct lined with respiratory epithelium. As this duct grows, it passes through the area in which the hyoid bone later develops. Therefore, in later life if the duct persists, it appears to pass through the hyoid bone. The thyroglossal duct is thus divided into two parts when the hyoid develops at the end of the second month.[9]

The duct begins to bifurcate forming the lateral lobes of the thyroid, the duct itself normally obliterated about the seventh week of fetal life. The point of origin of the duct is indicated by the foramen cecum, and the thyroid connection is represented by the pyramidal lobe of the thyroid.[10] If the growth of the thyroglossal duct is in any way arrested therefore, thyroid tissue may appear at any point of the tract of development of the duct. Thus we may have lingual thyroid or aberrant thyroid from the foramen cecum to the pyramidal lobe.[11, 12] Or, if the duct fails to obliterate, cysts may be found at any point along the tract. The most usual location of these cysts is between the level of the hyoid bone and the thyroid notch.

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The duct may persist merely as an opening at the foramen cecum periodically discharging a foul tasting material into the mouth. Sistrunk[17] suggests that infection may cause inflammation of the duct and the opening will be sealed off by edema and scar tissue. The normal products of the epithelial lining of the tract continue to be manufactured, and a thyroglossal duct cyst is formed. This may present itself as a bulging at the foramen cecum, or it may dissect along the tract to appear submentally. This undoubtedly accounts for some of the cysts, but it is probable that in some cases there is merely a small portion of the duct which does not obliterate and lies dormant for years until with adolescence it becomes activated, starts to secrete material, and forms a cyst. This cyst in turn may become secondarily infected.

Incidence. Thyroglossal duct cysts occur more often in men than in women. The average age of onset is 18 years, but the largest group appears before ten years of age. There was variation in appearance between five months and 77 years of age (Table 1).

History, Signs and Symptoms (Table 2). The main subjective complaint is swelling, usually recurrent, which appeared in 99% of cases. The symptoms had persisted from four days to 28 years before medical aid was sought. Infection was present in 66% on presentation. The usual history was of an upper respiratory infection followed by a midline tumor. However, 21% were lateral with the swelling more common on the left. Operation confirmed the diagnosis.

Twenty-five per cent of the patients had had previous attempts at removal, one patient having had seven such attempts before complete cure was obtained.

In 94% of cases a cyst was present with an accompanying draining sinus in 55%. The sinuses occurred in those patients who gave a history of infection with spontaneous rupture or attempts being made to incise and drain the lesion. There were several cases in which spontaneous rupture occurred with subsequent healing following the evacuation of the contents. In the draining sinuses the discharge was more copious in the presence of upper respiratory infections, and there were periods of complete closure with recurrence with the next throat infection. Less common complaints were difficulty in swallowing, fever, redness of skin, and hoarseness. In one patient there was complete inability to speak. In children dyspnea or respiratory obstruction may be the presenting complaint.

Physical Examination. The cyst may present itself as a localized swelling or as a diffuse phlegmon with involvement of the entire

TABLE 1.

AGE OF ONSET

AGE	NO. OF PATIENTS	%
1-10	45	35
11-20	27	21
21-30	17	13
31-40	18	14
41-50	12	10
51-60	6	5
61-70	1	1
71-80	1	1

TABLE 2.

HISTORY - SIGNS - SYMPTOMS

	NO. OF PATIENTS	%
Previous Operations—		
Attempts at Excision	33	25
Incision and Drainage	24	20
Infected	85	66
History of Upper Respiratory Infection	55	43
Swelling	127	99
Tenderness	42	32
Difficulty in Swallowing	24	20
Redness of Skin	19	15
Hoarseness	6	5
Fever	3	2
Difficulty in Phonation	3	2
Cyst Present	120	94
Fistula Present	70	55

TABLE 3.

BACTERIOLOGY

	No Growth	Staphylococcus Albus	Staphylococcus Aureus	Streptococcus Alpha-hem.	Streptococcus Beta-hem.	Streptococcus non-hem.	Pneumococcus	Influenza	Diphtheroid
1937-1941	5	7	3				1		
1942-1946	3	5	3	1	1	2		1	1
1947-1949	2	3 (coag.−)	1 (coag.+)	2		2	1	1	

TABLE 4.

TREATMENT

	NO. OF PATIENTS	% BASED ON 121 PTS. OPERATED ON
Surgical Excision	121	94
Hyoid Removed	90	74
Connections to Mouth Floor	87	72
Drain Used (without penicillin)	51	42
Drain Used (with penicillin)	0	0

neck. The foramen cecum may or may not be visible. If there is drainage into the oral cavity, the foramen cecum may be seen as a pin-point opening in the midline at the base of the tongue exuding white material. The mass may present itself in the mouth, in the submental region, or subhyoid region (the usual case) and is usually in the midline. The size varies from 2 mm to 7 cm in diameter. The skin is freely movable over the cyst, except in cases of secondary infection or fistula tract. The cyst moves on swallowing and is usually not tender on palpation. Seldom is the mass fluctuant. Indeed, the walls are usually swollen so tightly that they feel hard rather than cystic. The mass will transilluminate. The thyroid gland should be freely palpable as a separate entity from the cyst.

Bacteriology. In this series specimens from 46 cases were cultured. Twenty-two per cent showed no growth and 33% showed *Staphylococcus albus.* In those cases in which both throat and cyst cultures were taken, the organisms were usually the same. In several cases, tuberculosis was suspected; but no positive cultures were obtained. One case presented itself as a severe diffuse cellulitis of the

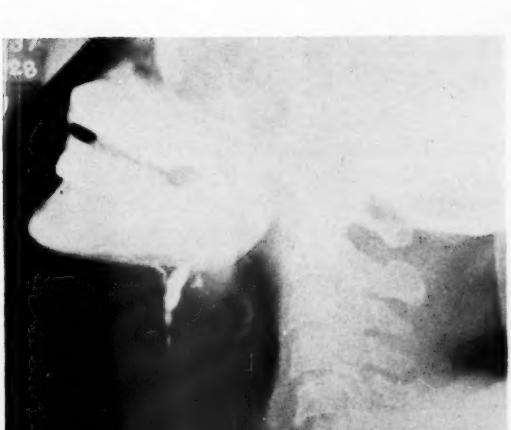

Fig. 1.—X-ray film illustrating the thyroglossal duct. (Courtesy of Dr. Alexander S. MacMillan.)

neck caused by hemolytic streptococcus and coagulase positive staphylococcus (Table 3). When the acute infection regressed, a thyroglossal duct cyst was found.

Diagnosis. The diagnosis is usually made without difficulty; however, several methods have been used to confirm the diagnosis in doubtful cases. In draining fistula it was found useful at times to instill lipiodol into the sac to see its ramifications. Dr. Alexander S. MacMillan at the Massachusetts Eye and Ear Infirmary has kindly presented a beautiful example of such a demonstration (Fig. 1). The tongue connections may be seen and the outline of the sac clearly followed through the hyoid.

Bailey[13] advocates paracentesis of branchial cysts and examination of the contents for cholesterol crystals. This was not done in any of this series, but in certain cases this diagnostic point may prove useful.[14]

The differential diagnosis may include almost any of the causes of swelling of the neck as reviewed by Lahey.[15]

Pathology. Gross: The cysts vary in size from 2 mm to 7 cm in diameter. They are filled with either a watery serous or thick purulent material depending on the amount of infection. Occa-

sionally a blood-tinged fluid is present. They are smooth, translucent, thin-walled cysts and are seldom multilocular. The cyst wall in one case was 5 mm thick, but as a rule the wall is very thin and easily broken.

Microscopic: Often on section the cyst cannot be demonstrated, and only a fistulous tract composed of fibrous tissue can be found. The cysts are originally lined with ciliated columnar epithelium, but as the pressure within the cyst is built up, the epithelium undergoes metaplasia to low cuboidal or squamous epithelium. This is the lining usually seen on pathological examination. Almost invariably there is chronic inflammation around the cyst. Occasionally typical thyroid tissue is found in the walls of the cyst. One case showed malignant degeneration of the duct remnant, with a pathological report of epidermoid carcinoma Grade II. Still another case showed typical tuberculosis lesions in spite of repeated studies for tuberculosis pre-operatively.

Treatment. The accepted treatment for thyroglossal cysts is complete surgical excision (Table 4). Some advocate attempts at obliteration with sclerosing solutions or x-ray,[16] but these procedures were not attempted in this series.

Although incision and drainage have their place in treatment, such a procedure should not be considered as sufficient. There are three indications for incision and drainage instead of excision.

1. Excision should never be done before the age of six years. Drainage and palliative treatment should be the rule before this age.

2. In the acute infectious phase with fluctuation, drainage should be done and the patient brought back several weeks later, after the acute flare-up subsides, for excision.

3. If the patient is too old, sick, or refuses to have excision, incision and drainage may be done to relieve swelling.

Chemotherapy has not changed the need for removal of the cyst. However, it has decreased the danger from secondary infection and lessens the postoperative hospital stay.

The anesthetic of choice is intratracheal ether, but in selected cases local anesthesia was used. Penicillin is given routinely in most cases and the average hospital stay postoperatively is five days.

The operative technique used by most of the surgeons was that suggested by Sistrunk[17] in 1920 (Fig. 2). He did not believe it practical to follow the tract from the hyoid bone up as it is friable and easily lost. He suggested "coring out" the duct by dissecting

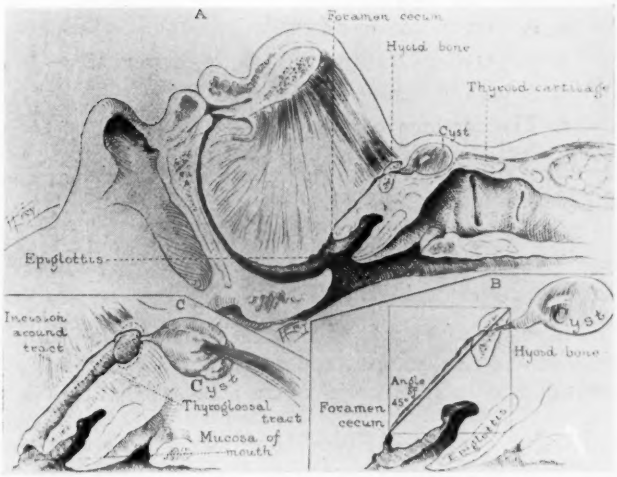

Fig. 2.—*A.* Sagittal section of the head giving the usual direction of the thyroglossal tract. The cyst is shown presenting between the hyoid bone and the thyroid cartilage. *B.* Dissection of the duct to be made along an imaginary line drawn at an angle of 45° from the intersection of lines drawn horizontal and perpendicular to the middle of the anterior superior portion of the hyoid. *C.* The duct with muscles surrounding it being "cored out" along the line shown. (From Sistrunk, Walter Ellis: The Surgical Treatment of Cysts of the Thyroglossal Tract, Ann. Surg. 71:121, 1920.)

for one-eighth of an inch on all sides in a line drawn at a 45° angle from the upper surface of the center of the hyoid bone in the midline of the neck, backward and upward toward the base of the tongue.

The complete Sistrunk operation may be divided into six steps.

1. Make a transverse incision two inches in length across the neck at the level of the hyoid.

2. Dissect the cyst to the hyoid bone. The tract usually passes through the hyoid.

3. Separate the muscles attached to the center of the hyoid and remove a portion of the bone, about one-fourth of an inch in length.

4. Without any attempt to isolate the duct, core through to the foramen cecum removing one-eighth of an inch of tissue on every side.

Finally removed will be:

a. A part of the hyoid
b. A portion of raphe of mylohyoid muscles
c. A part of genioglossus muscle
d. The foramen cecum

5. Close the opening into the mouth and draw the sections of the genioglossus muscle together.

6. Join the tissues surrounding the cut ends of the hyoid with chromic catgut to approximate the bare edges.

The injection of methylene blue into the tract to allow easier following of the ramifications appears frequently in the literature but it was done only in two cases of the entire 128.

Follow-Up. It was not possible to follow the entire group of cases as closely as desired. Exact conclusions as to the results of the operative procedures cannot be made in our series. However, many authors report 100% cures by use of the Sistrunk operation.

Several interesting facts were observed in 30 cases which were personally examined. There were two recurrences in this group. One patient had persistent discharge from the foramen cecum; and the other had a discharge from the lateral border of the incision, externally. In both of these cases the hyoid bone had not been removed. Neither patient thought the drainage severe enough to warrant further surgery.

One 36-year-old white female was found to have a firm, hard mass, 2 x 3 cm, at the base of the tongue. It had not been present on the previous admission. There were no subjective complaints but the patient was brought into the hospital for study. (Dr. LeRoy Schall diagnosed the mass as lingual thyroid.) Thirteen years previously the patient had had the successful removal of a thyroglossal cyst, this being confirmed microscopically. There was also a report of thyroid tissue in the specimen. The basal metabolic rate on this admission was -21 and a biopsy confirmed Dr. Schall's diagnosis. It was concluded that the original operation had actually removed a necessary portion of the patient's thyroid which was aberrant; and there was a resulting hyperplasia about the foramen cecum of the remaining thyroid tissue. The presence of thyroid tissue in the normal position should be confirmed before operation. Radioactive iodine and basal metabolic rate studies should be done in selected cases.

In those cases in which the incisions were (as advocated by Sistrunk) over or above the hyoid, there was considerable cicatricial tissue with wide deforming scars. Where the incisions had been made below the thyroid notch, they had healed with little scar formation and were barely visible.

SUMMARY AND CONCLUSIONS

1. A study of 128 cases of thyroglossal duct cysts is made with a review of the embryology.

2. Findings are considered under incidence, history, signs and symptoms, physical examinations, bacteriology, diagnosis, pathology, treatment, and follow-up.

3. The diagnosis of thyroglossal cyst should be considered in all upper midline and lateral swellings of the neck (21% in our series were lateral).

4. The cysts should be removed by the Sistrunk method, but palliative treatment should be the rule before six years of age, in the acute stage of inflammation, and in the old, poor operation risks.

5. Incisions lower than that advised by Sistrunk give better cosmetic results.

6. The presence of thyroid tissue in the normal position should be ascertained before removal of the cyst is carried out.

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REFERENCES

1. Rucker, Wm. H.: Congenital Cysts and Fistulas of the Neck, Minnesota Med. 31:413-417 (Apr.) 1948.

2. Gray, Henry: Anatomy of the Human Body, Philadelphia, Lea and Febiger, 1943, p. 59.

3. Meyer, Herbert: Congenital Cysts and Fistulae of Neck, Ann. Surg. 71: 121, 1932.

4. Hyndman, O., and Light, George: The Branchial Apparatus, Arch. Surg. 19:410 (Sept.) 1929.

5. Willy, Herbert: True Branchiogenic Cyst and Fistula of the Neck, Arch. Surg. 35:766, 1937.

6. Neal, H. B., and Pemberton, J. de J.: Lateral Cervical (Branchial) Cysts and Fistulas; Clinical and Pathologic Study, Surgery 18:267-286 (Sept.) 1945.

7. Anderson, W. A. D.: Pathology, St. Louis, C. V. Mosby Co., 1948, p. 1055.

8. Blassingame, C. D.: Congenital Cysts and Fistulas about the Head and Neck, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 56:395-403 (June) 1947.

9. Peterson, Edward J.: Tumors of the Neck, Am. J. Surg. 61:350-359, 1943.

10. Bolyeat, Fred: Cysts and Fistulas of the Branchial and Thyroglossal Ducts, J. Oral Surg. 6:60-64, 1948.

11. Wapshaw, H.: Lingual Thyroid, Brit. J. Surg. 30:160, 1942.

12. Goetsch, E.: Lingual Goitre, 3 Cases, Ann. Surg. 127:291-316 (Feb.) 1948.

13. Baily, Hamilton: The Diagnosis of Branchial Cyst, Brit. M. J. 1:940-941, 1928.

14. Hubert, L.: Thyroglossal Cysts and Sinuses, Analysis of 43 Cases, Arch. Otolaryng. 45:105-111 (Jan.) 1947.

15. Lahey, Frank H.: Tumors of the Neck, J. A. M. A. 138:264, 1948.

16. Cutler, E. C., and Zollinger, R.: Sclerosing Solutions, Treatment of Cysts and Fistulas, Ann. Surg. 19:411 (Mar.) 1933.

17. Sistrunk, Walter Ellis: The Surgical Treatment of Cysts of the Thyroglossal Tract, Ann. Surg. 71:121, 1920.

XXXIII

THE MALOCCLUSION FACTOR IN OBSTRUCTION OF THE EUSTACHIAN TUBE

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That there is a relationship between eustachian tube obstruction and dental malocclusion is well known. The purpose of this paper is to outline the method used in treatment.

Obstruction of the eustachian tube due to this factor is the culmination of a series of events starting with injury arising from malocclusion, creating a protective reflex with inhibition of muscular action in chewing and swallowing, which in turn not only may inhibit spontaneous opening of the tube and ventilation of the tympanic cavity, but may create a congestive swelling of the mucosa of the eustachian tube. Obstruction of the eustachian tube may be relieved by restoring the muscle action in chewing and swallowing.

The noted anthropologist Hooton considers that malocclusion is essentially a degenerative trend in our modern civilization, and that it is definitely on the increase to an alarming extent. He considers the easy availability of modern soft cooked foods, requiring the minimum exercise of the jaws and teeth, as one more potent factor added to an already obsolete biting and chewing mechanism.

There are several types of malocclusion and many variations which are capable of creating mechanical injury to the tissues during mastication. The injury is mechanical and may be limited to cuspal interference—even to a single set of opposing cusps. For instance, the mandible with its dental complement, in order to reduce injurious mechanical stimuli, spontaneously seeks a more favorable position from which to posture during the resting phase and to function during the chewing, even at the added expense of energy in maintaining malposture and performing malfunction. This would appear to be insignificant but when it is repeated innumerable times by reflex neuromuscular mechanisms throughout the entire skeletal system its cumulative effect is vastly greater than the local effect

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Read before the New England Otolaryngological Society meeting held at the Massachusetts Eye and Ear Infirmary, Boston, Massachusetts, November 17, 1948.

would suggest.[2] This may explain the easy fatigability in cases of malocclusion.

On the other hand, during mastication there may be multiple mechanical injuries transmitted through opposing teeth, which are out of axial alignment, to the alveolar structures, producing mechanical pyorrhea due to compression, shearing and torsional forces.

Loss of molar teeth on one side develops habitual chewing on the opposite side tending to overuse and to imbalanced mechanical forces in one or both temporomandibular joints.

In cases where there is extensive loss of lower molar support on both sides, with only the front teeth remaining to contact the upper teeth, the condyles of the mandible are forced into the glenoid cavity compressing the meniscus and lining membranes of the joints causing inevitable injury and destruction.

The temporomandibular joints become weight bearing joints, a function they were never intended to perform. They normally are toggle joints, designed for freedom of motion in mastication.

Not uncommon is the protrusive type in which the mandible is postured forward to the extent that the lower incisors are anterior to the upper incisors producing a forward thrust of the mandibular condyle against the tubercular eminence on the anterior surface of the glenoid cavity.

The open bite is another type in which there is an elliptical opening between the upper and lower incisors with the tip of the tongue filling the defect.

A frequent form of acquired malocclusion is the retrusive type with closed bite, in which the mandible is displaced downward and backward and the upper incisor teeth overlap the lower. The lower incisor teeth may contact the upper incisors on their lingual surfaces forcing them outward by a shearing force. Again in the more extreme cases, the mandible has retruded to the extent that the lower incisors may contact the necks of the upper incisors or even more posteriorly the mucous membrane of the hard palate.

This type of malocclusion favors a corresponding displacement of the hyoid bone with the base of the tongue in a downward and backward direction, such as to set up a potential threat to the vital airway of the hypopharynx. This threat is counteracted by the "safety muscles" of the pharyngeal airway. They are the geniohyoid and genioglossal muscles. They posture the hyoid and base of the tongue upward and forward.

This may account for the hypertension found in the muscles of this region and the associated hyperactive gag reflex, both of which may create an inhibition of the other allied muscle groups.

Let us consider now the protective reflex with its powerful over-all inhibitory influence.

Under conditions of malocclusion when the powerful forces of compression, torsion and shearing develop mechanical stimuli of such intensity that they are injurious to the tissues, the receptors for injury take over and there is created the nociceptive reflex of Sherrington.[1, 2]

The purpose of the nociceptive reflex is protection of the tissues against a repetition of injury and it will persist as long as injury persists or is imminent. The inhibitory pattern frequently becomes habitual, persisting after the initial phase has ceased to exist especially in the apprehensive individual.

It is essential to appreciate the fact that not all injurious stimuli register as pain.[2, 3] During the brief acute phases of malocclusion, pain may be experienced but during the long intervals of quiescence stimuli can still be injurious to the tissues but not of sufficient intensity to register as pain. Pain is a psychic experience and when present may be a powerful influence in reinforcing the inhibition pattern. Pain memory patterns may persist and involuntarily reinforce and perpetuate the protective reflex during the long quiescent periods.

Under control of the protective reflex the mandible, since it is the movable part, will be restricted both as to motion and force by virtue of the increased tension of the muscles of mastication. It amounts to a partial trismus. Nature in her wisdom is using the jaw muscles to splint the parts against further injury to teeth and their supporting structures, namely, the alveolar ridges and sockets, the gums, periosteum, periodontal membrane and temporomandibular joint structures.

An analogous example is seen in baseball when the batsman in bunting the ball shortens up on the bat for the sake of control of force and accuracy of direction.

Let us turn now to a consideration of the eustachian tube and the nasopharynx. Under normal conditions of occlusion the chewing reflex, by its own activity generates stimuli which spread to the neuromuscular elements of the next reflex which is to follow, namely, the swallowing reflex. By means of the excitatory spread of one reflex to the next about to go into action, a smooth, integrated and co-ordinated action between allied groups of muscles is possible.

On the other hand, if the chewing reflex is inhibited there will be an inhibitory spread to the swallowing reflex. This means, then, that the swallowing reflex will be inhibited, therefore the voluntary muscles which initiate the first act of swallowing, namely, the tubopalatal and pharyngeal muscles including the superior pharyngeal constrictors, will be inhibited.

Regarding the capillary flow of blood and lymph in the vascular beds of the nasopharynx and the eustachian tube, it is known that the lymphatic flow from the tympanic cavity is by way of the lymphatics of the eustachian tube and from these along the lymphatics of the lateral wall of the nasopharynx to the lateral pharyngeal lymph glands located in the buccopharyngeal fascia.

It is known that the lymphatic flow of the nasopharynx is influenced by the contractile activity of the superior pharyngeal constrictor muscles, which, with the buccopharyngeal fascia, form the lateral walls of the nasopharynx. Since it is a well accepted fact that muscular contraction increases the capillary flow of blood and lymph, it is reasonable to assume that inhibition of muscular activity will be accompanied by a diminished flow, even to favoring a local stasis.

The result is a diminished blood and lymph flow of the vascular bed of the lateral walls of the nasopharynx and the mucosa of the eustachian tube, in addition to inhibition of auto-inflation of the tympanic cavity.

Such a combination of factors may explain certain cases of eustachian tube block in which a funnel shaped mucus plug has formed in the osseous portion of the tube, making inflation difficult or impossible until a paracentesis is done. Then, the inflation is easily performed, the mucus removed and all goes well until the opening in the drum closes, when the condition recurs. There is still another type of case in which the patient is very grateful for the relief afforded by inflation, only to report that it was of very brief duration and repetition of inflation is valueless.

Patients with loose artificial dentures are markedly inhibited in muscular activity due to the dominant necessity of retaining them within their mouths, to say nothing of keeping them in occlusal relation. Muscular exercises combined with improving the fit of the plate or plates, will frequently relieve the eustachian tube block to a surprising degree. Many patients, for the first time, learn to control their plates by neuromuscular exercises and with marked benefit to the eustachian tube block.

It would appear that World War II has demonstrated the necessity of seriously studying malocclusion, and an excellent beginning has been made. Two dentists, Kelley and Langheinz, assigned by the U. S. Navy Department to study dental malocclusion under extremes of atmospheric pressure changes in submarine personnel at New London presented a report which was of particular personal interest.

They sensed in part the neuromuscular explanation given by the writer in two previous papers[1, 2] but failed to appreciate that changes in the lymphatic flow is secondary to muscular action, as given in detail in this paper.

In the meanwhile the neuromuscular exercises about to be presented have been devised or borrowed by the writer to meet the needs of the individual case and have proved to be of very real value in rehabilitating a more normal chewing and swallowing muscular activity, and diminishing the eustachian tube block with a better hearing function. The keynote struck by the writer during the past 12 years has been the chewing reflex, and within the past year this basic guide has been corroborated by Froeschels, who, since 1933, has utilized the primitive chewing reflex in speech therapy.[4] He told me that he uses it now for controlling stutterers and stammerers by having them start chewing their breath and the words they wish to enunciate, with the result that the desired speech flows out naturally. His concept that chewing is the basis of speech is based upon the observation of a Dutch trader, who, in approaching a primitive village of a North African tribe was unable to tell from the sounds whether they were eating or holding a pow-wow. He found that they were merely eating in their usual manner.

Brueckner, a noted South African missionary, told me that the Zulus follow the same technique and that their voices readily carry an incredibly long distance. He speaks Zulu fluently and gave me a demonstration of their speech which corroborated the trader's observations and Froeschels' basic concept.

Regarding the neuromuscular exercises, it should be stated that they are based upon the principle of reconditioning each component muscular group of the chewing and swallowing reflex, in order that they may measure up to their fullest individual functional ability, and that as members of a team they are capable and ready to respond in a smooth, rhythmic, co-ordinated manner.

The component groups of the chewing and swallowing reflex are the mandibular (per se), mandibulofacial, mandibulolingual,

mandibulohyoid (supra and infra), mandibulotubopalatal, mandibulopharyngeal, notably the superior constrictors of the nasopharynx.

The primitive chewing reflex advocated by Froeschels, in speech therapy, has been added during the past year, since it combines all the indicated separate exercises into one exercise designed for team play.

The principle of exaggerated slow motion in muscular exercise, is to emphasize the reawakened kinesthetic pattern to the degree that it will overcome the habitual old patterns. Volition is also a powerful factor, especially when guided by the mirror held in one hand while palpating the movements of the hyoid bone with the other.

SUMMARY

A working hypothesis is presented which assumes that there is a relationship between eustachian tube block and dental malocclusion due to mechanical injury creating a protective reflex with inhibition of the normal muscular action of chewing and swallowing, which in turn not only inhibits auto-inflation of the tympanic cavity, but creates a local congestive swelling of the mucosa of the eustachian tube. Neuromuscular exercises to counteract the muscular inhibition have been found to be capable of reversing the changes producing eustachian obstruction.

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REFERENCES

1. Seaver, Edwin P., Jr.: Temporo-mandibular Joint Malocclusion and the Inner Ear—A Neuromuscular Explanation, Annals of Otology, Rhinology and Laryngology 46:140 (Mar.) 1937.

2. Seaver, Edwin P., Jr.: The Neuromuscular Control of the Mandible, Am. J. Orthod. and Oral Surg. 28:222-229 (Apr.) 1942.

3. Seaver, Edwin P., Jr.: Fear and Pain in Otolaryngological Surgery, Eye, Ear, Nose and Throat Month. 17:94-97 (Apr.) 1938.

4. Froeschels, Emil: Hygiene of the Voice, Editorial, J. A. M. A. 135:1154 (Dec. 27) 1947.

NEUROMUSCULAR EXERCISES FOR MALOCCLUSION

GENERAL INSTRUCTIONS:

1. Hold mirror in one hand and hyoid bone in the other for guidance and reinforcement.

2. Tilt head up rather than down as the eustachian tube opens to less pressure when head is up. This is also of mechanical advantage to hyoid musculature. Use correct bodily posture when sitting down.

3. Loosen tight collars or neckwear which may be impeding the infrahyoid muscles.

4. Perform each exercise three times slowly, not rapidly, similar to stretching and contracting one's arms on awakening in the morning to feel out the muscles and increase their tone.

5. Allow a rest interval between exercises. Usually 5 to 10 seconds is sufficient for advantageous recovery, but a longer interval in elderly patients is advisable.

6. Exercise three times daily at the start and increase the frequency to establish the habit.

It is advisable to start the patient with one group of exercises at a time. After checking on subsequent visits to see that they are being carried out adequately the next group can be added.

Check the exercises at each visit before inflating the ears. The "warming up" preliminary to inflation secures better co-operation.

SPECIFIC INSTRUCTIONS:

MANDIBULOFACIAL EXERCISES

1. Enunciate the vowels A, E, I, O, U. Each vowel is to be enunciated slowly and with greatly exaggerated expression.

2. Enunciate vigorously Ki-Ki-Ki, Kah-Kah-Kah, I-I-I.

MANDIBULOLINGUAL EXERCISES

1. Practice straight protrusion and retrusion of tongue alternately with mouth held wide open throughout the exercise. During retrusion let the tip of the tongue rest on the floor of the mouth while the base of the tongue is pulled well down.

2. Protrude the tongue and slowly rotate clockwise while holding the mouth wide open.

3. Repeat counter-clockwise.

MANDIBULOHYOID EXERCISES

1. Place the tip of the tongue against the roof of the mouth for support while alternately contracting and relaxing the anterior neck muscles. The fingers of one hand should be made to palpate the hyoid bone and the contracting hyoid muscles for the sake of reinforcement and appreciation.

2. Brace the tip of the tongue against the lower incisors, and forcibly rotate the hyoid and the base of the tongue forward by alternately contracting and relaxing the anterior neck muscles.

3. Open the mouth wide, place tip of tongue behind the upper incisors and firmly wipe the roof of the mouth backward in an attempt to touch the uvula.

MANDIBULOTUBOPALATAL EXERCISES

Hold the mouth wide open, hold down the base and the tip of the tongue firmly flattened on the floor of the mouth while alternately contracting and relaxing the soft palate. The soft palate should be elevated to shut off the nasopharynx. The uvula even of the flaccid elongated type will be seen to respond. As the musculature improves in function, some uvulae may be seen almost to disappear. During this exercise patients may be conscious of autoinflation of the middle ear.

MANDIBULAR EXERCISES

Practice primitive type chewing with a forcible swallowing act at favorable intervals. It will be noted that the jaw opening and closing muscles have been an integral part of all the exercises which are in reality based upon individualizing each component of the chewing and swallowing reflex.

Chewing on a large bolus of gum has been found to be of practical value.

XXXIV

DETERMINATION OF SUSCEPTIBILITY TO ABNORMAL AUDITORY FATIGUE

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The prevention of traumatic deafness lies, to a large extent, in a better understanding of the phenomenon of acoustic trauma. The relation between abnormal auditory fatigue and the irreversible loss of hearing caused by acoustic trauma has been demonstrated in previous experimental studies.[1, 2] Particularly stressed was the practical application of this correlation in determining in advance persons who may be unduly susceptible to acoustic trauma.

It is becoming increasingly apparent from the vast flood of reports in the literature that we have failed to solve the problem of traumatic hearing loss. Granted that no known form of therapy can improve this type of hearing defect, it becomes clear that energy must be directed toward the prevention of deafness in persons not yet affected and the arrest of progress in those who have already suffered some loss.

Much work has been done on ear protectors. From the first report of the use of cotton in the ears by Chippendale[3] in 1866 to the recent description of a modern neoprene ear plug by Weiss,[4] attempts have been made to filter out harmful noise. As far back as 1890, Cousins[5] recommended the use of ear plugs made of India rubber. The most that has ever been accomplished is the lessening of noise by 25 or 30 db. This affords adequate protection for moderately intense noise levels. As the decibel reading for noises known to cause acoustic trauma lies between 80 and 90,[6] it is apparent that intensities over 110 db are still capable of causing permanent cochlear damage, even though ear protectors are worn.

The unpopularity of ear protectors among those employed in noisy industry or in the armed forces is well known. They are both uncomfortable and inconvenient. Even when furnished with such devices most men discard them after a short time. They make difficult the hearing of orders by those in the armed forces. In industry

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Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.

an increased accident hazard is afforded through the inability of men with plugged ears to hear warnings of impending danger given by fellow-workers.

In susceptible individuals acoustic trauma may be caused by a total by-passing of the air conduction route. Floor and machinery vibration may be carried to the cochlea directly through bone conduction from skeletal contact with a vibrating part.

Aside from the elimination of noise at its source, which seems to lag far behind the creation of new, noisier machines, the most practical approach to the problem appears to be in preventing the actual exposure of those most susceptible to acoustic trauma. This is impractical in time of war, but in peacetime drafts, and particularly in industry, individuals predisposed to abnormal aural fatigue and irreversible traumatic loss should be eliminated from noisy occupations.

That this group may be spotted by a method of fatiguing as described below has been previously demonstrated. Until state legislative bodies rule traumatic deafness a compensable occupational disease, little will be done. When paid compensation is higher than the cost of such pre-employment testing, the economy of such a program may find popular appeal.

Granted that vulnerability to acoustic trauma and undue auditory fatigue are both abnormal conditions, it remains to be determined just what shall be classified as abnormal. Ewing and Littler[7] noted that the conclusions of early workers on auditory fatigue were of a somewhat inconsistent character. They were found to agree that large individual differences are seen as between different subjects and even as between the two ears of one subject. This pronounced individual variation in susceptibility has been noted and stressed by many authors.[4, 7-11]

Smith,[12] after working with human subjects, theorized that it is the lengthening of the refractory period of the individual nerve fibers by the extreme intensity that causes fatigue. This increased refractory period results in a relative decrease in the number of working fibers at any particular moment, with a consequent decrease in the sensitivity itself. In a later work he[13] advanced the theory of a dual origin of auditory fatigue, classifying these as peripheral or cochlear and central or cortical. Either may appear alone or both together in varying amounts in the stimulated ear. In the contralateral ear, however, only the central effect can produce the threshold rise.

Kobrak, Lindsay and Perlman[14] divided the phenomenon of auditory fatigue into three different types. The first or central type was demonstrable in individuals showing subjective fatigue without signs of diminished cochlear irritation. The second or peripheral type was demonstrable in those showing both objective and subjective elevation in auditory threshold. The third was known as auditory adaptation and was likened to the light-dark adaptation of the eye. Here was demonstrated a protective mechanism capable of decreasing the sensitivity of the ear during prolonged stimuli, quite apart from the protection from sudden stimuli afforded by the muscle reflexes of the middle ear. This phenomenon was differentiated from the first two types in that its effect is lost immediately upon cessation of the stimulating tone.

EXPERIMENTAL STUDY

Method. The present study was made on both enlisted men and officers who presented themselves as patients or for routine examination at the ear, nose and throat clinic of an A.S.F. Regional Hospital. In no instance were subjects chosen who gave complaints referable to their ears or in whom nose and throat pathology was present that might conceivably affect hearing acuity.

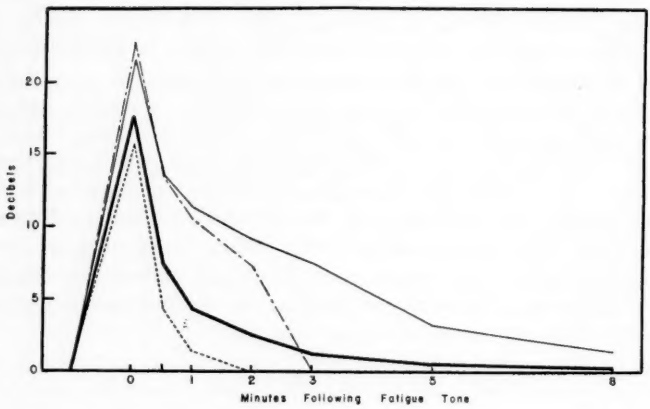
Only intelligent, co-operative subjects were chosen for the series. Any subject who failed to uphold this criteria by his responses during the procedure was dismissed without further testing. Men whose hearing threshold at 4096 cycles was higher than 30 db in either ear were not included in the series.

Each subject was questioned with regard to deafness in his family and whether he had at any time noted tinnitus. Following the completion of the test he was questioned concerning the occurrence of tinnitus on cessation of the fatiguing tone.

A Maico D 6 audiometer, just calibrated by the manufacturer, was used in this study. A soundproof room was available. The test was explained to each subject. He was then acquainted with the audiometer by determining several trial frequency thresholds.

His hearing threshold at 4096 cycles was then determined for each ear. Readings were recorded at 5-db intervals.

Following the threshold determination, one ear was then exposed to a 2048-cycle fatiguing tone at an intensity of 80 decibels for a five-minute period. Immediately upon cessation of the fatiguing tone the threshold for the same ear at 4096 cycles was taken and recorded. One-half minute following cessation of the tone the threshold was again taken. Readings were continued at one minute, two



Mean threshold rise at 4096 d.v. following exposure to 2048 d.v. at 80 db for five minutes.

minutes, three minutes, five minutes and eight minutes following cessation of the tone, or until the threshold difference became less than 10 db from the predetermined normal for that ear. A deviation of less than 10 db was considered to be within experimental error.

The opposite ear was then fatigued and tested in the same manner. A consistent threshold following termination of the fatiguing tone was generally obtained in less than ten seconds from the given testing time and always within 15 seconds.

Observations. The average age of the 250 subjects was 26.6 years. Thirty-two men gave a history of deafness in the family. In each instance, however, but one member was affected. There was no appreciable difference in the responses of these men to the fatiguing tests and the responses of the entire series. Ten soldiers gave a history of previous attacks of tinnitus of consequence.

All men tested were classified in one of three groups according to the time lapse necessary for recovery from auditory fatigue. In the first group (a) were placed all subjects who showed fatigue in one or both ears after three or more minutes time lapse following cessation of the fatiguing tone. In the second group (b) were placed all subjects who showed fatigue in one or both ears after a

two-minute time lapse following cessation of the fatiguing tone, but who showed no fatigue at the three-minute testing period. In the third group (c) were placed the remaining subjects, those who recovered from auditory fatigue in less than two minutes.

Of the 250 men exposed to the fatiguing tone, 40 fell in the first group (a), 37 in the second group (b) and 173 in the third group (c). Of the 40 men in the first group (a), 18 still showed fatigue after a five-minute time lapse and 9 had failed to recover upon termination of the test at eight minutes.

Thirty subjects experienced bilateral tinnitus following exposure to the 80-db tone. Of these, 12 fell in the first group (a), 6 in the second group (b) and 12 in the third group (c). Tinnitus in the right ear only was experienced by 28 men. Nine fell in the first group (a), 6 in the second group (b) and 13 in the third group (c). Tinnitus in the left ear only was experienced by 20 men. Four fell in the first group (a), 3 in the second group (b) and 13 in the third group (c).

COMMENT

The wide variability in the rapidity with which the human ear recovers from aural fatigue in different individuals is demonstrated by the accompanying chart. Kobrak, Lindsay and Perlman[14] pointed out that acoustic fatigue is a protective mechanism for decreasing the sensitivity of the ear during prolonged loud sounds. It must be recognized that this is normal and that abnormality is reached only when a prolonged recovery time is encountered.

An attempt was made in this study to classify the 250 subjects according to what was considered to be normal or abnormal auditory fatigue. It soon became apparent that no definite line could be drawn separating the normal from the abnormal. Therefore, a borderline or intermediate group was formed. The response necessary to be classified in any one group has been outlined above.

The 40 subjects showing auditory fatigue following a 3-minute time lapse were considered to be definitely abnormal in their delayed recovery. The 37 subjects showing fatigue at the end of 2 minutes but recovering by 3 minutes were considered to be slow in their ability to recover but not alarmingly abnormal. The 173 subjects recovering from all auditory fatigue in less than 2 minutes were considered normal in their response.

Mention should be made of the number of subjects who complained of tinnitus following discontinuance of the fatiguing tone who, after a two to three-minute time lapse, would volunteer that

the tinnitus had suddenly ceased. The majority would then show a return of hearing threshold to normal at the next determination time. Many of these subjects fell into the second (b) group. The relatively sharp terminal drop of this group curve suggests the sudden termination of an interference factor.

The reasoning behind the selection of a 2048-cycle fatigue tone and a 4096-cycle testing frequency should be explained. Perlman[15] concluded that when a rise in threshold occurs after fatigue it generally reaches a maximum one octave above the fatigue tone when the latter is between 512 and 4096 cycles inclusive. This, and the established fact that the most vulnerable spot in the cochlea is found in the 4096 cycle area, led to the choice. A further advantage of 2048 cycles as a fatigue tone is seen when one considers Wiggers' observation[16] that conduction efficiency is appreciably decreased by intra-aural muscle action only below 1000 cycles. Previous studies[2] in which 2048 cycles was used as a fatigue tone followed by a determination of threshold at each octave bore out Perlman's observation.

In this series it is hoped that interest will be focused primarily on the abnormal fatigue group. The relationship between abnormal auditory fatigue and irreversible traumatic loss of hearing has been demonstrated in previous studies. Now a more complete picture of auditory fatigue is afforded. This makes possible a relatively accurate grouping of subjects according to response.

CONCLUSION

This series of tests suggests the following conclusion:

The ears of certain persons are more susceptible to auditory fatigue than are those of others. Ears in which prolonged fatigue is demonstrable are considered to be abnormal.

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REFERENCES

1. Wilson, W. H.: Prevention of Traumatic Deafness: A Preliminary Report, Arch. Otolaryng. 37:757-767 (June) 1943.

2. Wilson, W. H.: Prevention of Traumatic Deafness: Further Studies, Arch. Otolaryng. 40:52-59 (July) 1944.

3. Chippendale, J.: Deafness Produced by Reports of Firearms, Correspondence, Lancet 2:170 (Aug. 11) 1866.

4. Weiss, J. A.: Deafness Due to Acoustic Trauma in Warfare, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 56:175-186 (Mar.) 1947.

5. Cousins, W., in discussion of Barr, T.: Injurious Effects of Loud Noises on the Hearing, Brit. M. J. 2:675 (Sept. 20) 1890.

6. Bunch, C. C.: Conservation of Hearing in Industry, J. A. M. A. 118:588-593 (Feb. 21) 1942.

7. Ewing, A. W. G., and Littler, T. S.: Auditory Fatigue and Adaptation, Brit. J. Psychol. 25:284-307, 1934.

8. Cope, B. A., and Johnson, H. B.: Aural Scotomas Produced by Gunfire, Arch. Otolaryng. 45:676-686 (June) 1947.

9. Campbell, P. A.: Aviation Otology in World War II, Laryngoscope 54: 387-397 (Aug.) 1944.

10. Patt, H. M.: Auditory Acuity of Artillerymen and of Rifle Coaches, Arch. Otolaryng. 44:298-302 (Sept.) 1946.

11. Stewart, J. V., and Barrow, D. W.: Concussion Deafness, Arch. Otolaryng. 44:274-279 (Sept.) 1946.

12. Smith, A. F. R.: Auditory Fatigue, Brit. J. Psychol. 25:77-85 (July) 1934.

13. Smith, A. F. R.: Experimental Deafness, Brit. J. Psychol. 26:233-244 (Jan.) 1936.

14. Kobrak, H. G., Lindsay, J. R., and Perlman, H. B.: Experimental Observations on the Question of Auditory Fatigue, Laryngoscope 51:798-810 (Aug.) 1941.

15. Perlman, H. B.: Acoustic Trauma in Man, Arch. Otolaryng. 34:429-452 (Sept.) 1941.

16. Wiggers, H. C.: Functions of the Intra-Aural Muscles, Am. J. Physiol. 120:771-780 (Dec.) 1937.

Scientific Papers of the American Laryngological Association

XXXV

PRESIDENTIAL ADDRESS

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PORTLAND, ORE.

With a deepening sense of personal appreciation for the high honor of presiding over this seventy-first meeting of the American Laryngological Association, I am reminded that this is the first time we have come to the shores of the Pacific. The founders and early members of this Association lived and carried on their scientific work at a time when it took a couple of weeks to cross the continent; specialization had been frowned on, and contributions were relegated to general medical journals and often were published many months late in abstract form. Under such circumstances, the characteristic independence of thought and action of our members was demonstrated from the beginning. Although our Association arose among men from New York, Philadelphia, Boston and Baltimore, we should remember that numerous brilliant pioneers came in from the middle states—then called "West"—men like E. L. Shurly of Detroit, Ingals and Casselberry of Chicago, De Roaldes of New Orleans. Somewhat later came our first member from San Francisco, Henry Wagner—trained in Europe, and like others just mentioned, cosmopolitan in his reading and approach to rhinolaryngologic problems.

These somewhat isolated pioneers were not bound by precedents arising abroad or in the large clinical centers of northeastern cities; they worked along lines often original, sometimes repetitious owing to lack of medical library facilities; yet they built better than they knew, traveling year by year to the meetings of our Association, and working toward institutions which have grown with the communities they helped to form.

One finds much of value in running through old volumes of our Transactions, to ascertain what our founders thought when they were our age; what they hoped for and anticipated, and often brought about with relatively primitive equipment. For example, it is now more than fifty years since Henry Wagner maintained that the main defense of the upper respiratory tract lay in the protoplasmic activity of living body cells, and in the "protective enzymes" produced by circulating leukocytes. It took a long time for his theory to gain acceptance.

At three score and ten, men are not usually originators; valuable as teachers, for experience and counsel, their finest original thought came between thirty and fifty. The best-trained technician nowadays often lacks the background of general culture which our pioneers had acquired. Broader horizons and better understanding of our changing world are too often neglected by narrow specialization.

Typical of the scientist isolated far from those who later held him in highest repute was Santiago Ramón y Cajal, illustrious Spanish neuro-anatomist, author, teacher and Nobel prize winner. Instead of my own inexpert generalizations, permit me to translate for you some of his comments on the life and work of the medical scientist in our time:

"The human brain represents a world wherein some continents have been explored yet vast lands remain unknown. The common untutored man is ignorant of everything and does not at all suspect his potential riches. On the other hand, the cultivated man tries to explore himself, and finally succeeds in discovering some hidden treasures. Some few moreover, have through reflex attention and internal strength succeeded in purifying their mental geography. How many happy fancies still await us at the intersections of our cells and our nerve pathways, if we impose on ourselves the task of auto-observation, methodically and patiently, in the light of science, and the warmth of meditation!

"Lest posterity forget us, we should hasten to conquer the present. Thus it is well to set hands to the plow in the fullness of youth, before the chill of years diminishes vigor and allays enthusiasms. The important thing is to conquer a corner in people's souls and in books, where our ideas will be preserved; *in fine* to emerge from the anonymous mass numbered by millions, to enter in one's own right the brilliant legion who are counted one by one. Such

work is hard, and bitter the reverses and contraditions of the struggle; but how delightful is victory if it deigns to smile on us! . . .

"Let those truly enamored of science direct their spiritual attitudes toward originality, before the deforming pressure of their surroundings and the burdens and toils of family convert them, by sheer weight, into vulgar gold seekers. Only by working ourselves do we teach others how to work. . . .

"When we reach the icy summit of old age, we may take account that we have survived many successive existences, held together by the luminous thread of our conscious memory. Like the deposits of prehistoric caves, our memory contains various strata characterized by relics of human tribes which have successively disappeared. The discreet old person should gaze with regret at the savage predecessors in his cerebral cavern, and declare himself out of sympathy with their actions and thoughts. . . .

"When one considers the healthy color and calmness of soul of pious people, one must think that religion possesses in addition to its high moral value, excellent nutritive qualities. Faith strengthens, and is conducive to longevity, while doubt sometimes condemns one to suffering and premature old age. . . .

"We have faith in future science; and we may hope that superior culture and better knowledge of the intimate mechanisms of life may succeed in creating a stronger and more durable humanity. Certainly, before reaching such a high goal, a few stubborn and obstinate organs may protest, functioning as brakes. But venturesome times are coming, and equilibrium between the organism and the supreme demands of progress will bring about a magnificent reality. And man, we must believe though it be yet a dream, will live more and better in the future."

Ramón y Cajal, like our own Mosher, was many-sided in his activities and was not only eminent in his special field, but directed his attention to the arts, to literature, history, languages, and other humanistic pursuits. These leaders afford us an example by which, as we grow older, we may richly profit. Most of us are or have been teachers; and we should perhaps be reminded of the value of alternative interests and occupations, as stimulants to our flagging regard for the humdrum tasks of every day.

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XXXVI

AZYGOS LOBES OF THE LUNG, AND TERMINOLOGY OF THE BRONCHOPULMONARY SEGMENTS OF THE BRONCHIAL TREE

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A so-called azygos lobe is found as a mesial part of the right upper lobe in less than 1% of autopsies. The term "azygos" means unpaired or accessory, and is used in reference to veins such as the azygos, and to lobes (in reality segments or parts of the latter) of the lung.

In the upper lobe of the right lung it is the result of an anomaly in the development of the azygos vein, which normally takes origin from the lumbar veins, passes upwards by the side of the spine, and goes forward over the hilum or root of the lung to join the posterior aspect of the superior vena cava. This is well seen in an illustration in Callender's Surgical Anatomy.[1] When it deviates laterally, it cleaves the apical segment of the upper lobe.

We recently came across a case with an azygos lobe which we were able to dissect and inflate. On opening the upper lobe bronchus and the apical segment bronchus, the latter had four subsegmental orifices. Of the two upper orifices (Fig. 1), one, O.1.P.M., on inflation served the posterior part of the azygos "lobe," and the other, O.1.P.L., the posterior part of the remainder of the apical segment, which is on the lateral side of the cleft (Fig. 2). The two anterior orifices similarly inflated the anterior part of the azygos "lobe," and the anterior part of the apical segment on the lateral side of the cleft. The apical segmental bronchus in normal specimens has a short trunk which divides into two branches, ventral and dorsal, each of which divides into a lateral and a mesial branch. The cleft is caused by an anomalous azygos vein passing between these four latter branches.

As the so-called azygos lobe of the right upper lobe of the lung is simply the result of the cleaving of the apical segment of that

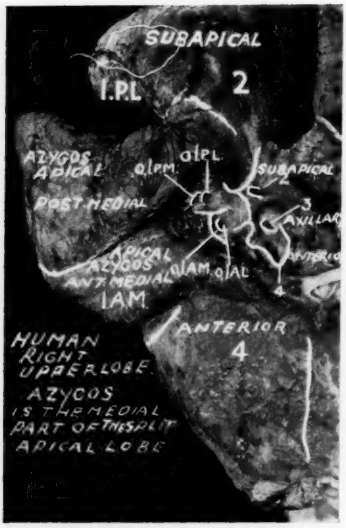

Fig. 1.—Mesial view of the right upper lobe. Apical segmental bronchus opened to show contained orifices. O=Orifice.

1. Apical segment.

1.P.M. Posterior part of azygos.

1.A.M. Anterior part of azygos.

O.1.P.M. Orifice leading to 1.P.M.

O.1.A.M. Orifice leading to 1.A.M.

O.1.P.L. Orifice leading to posterior part of apical segment on outer side of cleft.

O.1.A.L. Orifice leading to anterior part of apical segment on outer side of cleft.

2. Subapical segment.

3. Axillary or lateral segment.

4. Anterior segment.

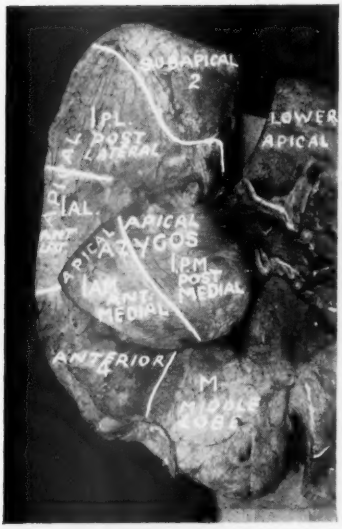

Fig. 2.—Azygos "lobe" drawn medially showing lateral and medial sides of the cleft caused by the azygos vein.

1.A.L. Anterior part of non-azygos part of the apical segment, lateral side of the cleft.

1.P.L. Posterior part of non-azygos part of apical segment.

lobe, the term is a misnomer. The term "azygos apical subsegment" or "azygos subsegment" would be more appropriate.

In the lower lobe of the right lung, a partially detached cardiac segment has at times evidently been considered as an azygos lobe. In our dissections of the human and the koala, we found that they were the only mammals with the cardiac segment normally incorporated with the lower lobe.[2] A partial detachment of some segment occurs about as commonly as the "azygos lobe" of the right upper lung.

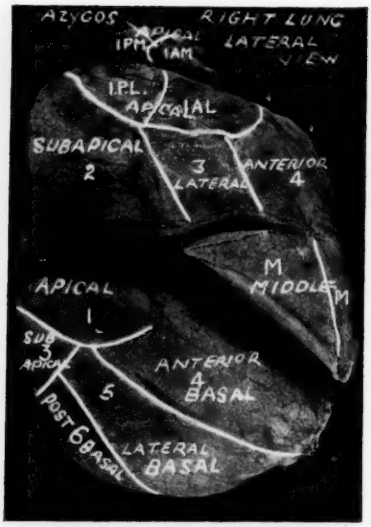

Fig. 3.—Lateral aspect of right lung with segmental boundaries delineated by inflation.

3. Axillary or lateral segment of upper lobe.

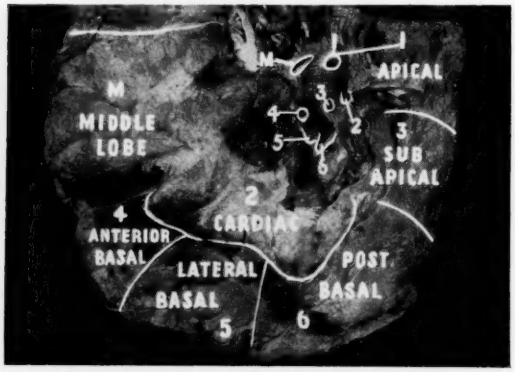

Fig. 4.—Mesial side of the lower lobe of the same lung, with segmental orifices shown and segments delineated by inflation. The cardiac shows a slight separation, also shown in the lower lobe left lung, in Fig. 8, p. 11, of reference 2.

The so-called azygos lobe of the left lung lobe is a partially separated part of the anterior basal segment. One speculative contributor thought it was a cardiac lobe of the left side. As a matter of fact, in gibbon and colobaye monkeys, which have a cardiac lobe on both sides, Dévé[3] has shown that on the left side it comes off as would be expected, just below the apical of the lower lobe, which is obviously well above the level of the anterior basal.

Figure 4 is an illustration of the lower lobe of the right lung of the specimen with the so-called "azygos lobe" discussed in this paper. The bronchial tree is opened to show the segmental orifices and the segmental areas. The cardiac segment stands out with a suggestion of a minor tendency to separation, more than is shown in the illustration.

At a meeting of otolaryngologists and chest surgeons in Honolulu at the Pan-Pacific Surgical Congress, 1948, a new terminology of the bronchopulmonary segments was approved as conforming to modern anatomical terminology. The diagrams with this paper may be altered accordingly. The term "axillary" has been eliminated to conform with modern terminology.

REVISED TERMINOLOGY OF THE BRONCHOPULMONARY SEGMENTS

Upper Lobe: Apical, lateral, anterior, subapical.

Middle Lobe: Anterior, lateral.

Lower Lobe: Apical, subapical, cardiac, anterior basal, lateral basal, posterior basal.

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REFERENCES

1. Callender, C. Latimer: Surgical Anatomy, Philadelphia, W. B. Saunders Co., 1933.

2. Hardie Neil, J., and Gilmour, W.: Anatomy of the Bronchial Tree, New Zealand M. J. 45:20-34 (Feb.) 1946.

3. Dévé, F.: Note complémentaire au sujet des lobes postérieurs et cardiaques due poumon, Bull. de la Soc. Anat. de Paris Ser. 6, 5:270-275 (Mar.) 1903. 1903.

XXXVII

THE NERVUS TERMINALIS

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As is well known, the olfactory membrane constitutes an area of yellowish color on the upper portion of the nasal septum and on the superior concha of the adjacent lateral wall of the nasal cavity. In man it is limited to about 500 sq. mm. in area, its boundaries being very irregular. In macrosmatic mammals, however, it is relatively much more extensive. The olfactory epithelium consists of olfactory cells, supporting cells, and basal cells. Beneath the epithelium are located the olfactory glands of Bowman. These glands are of branched tubuloalveolar type, the secreting tubules and alveoli lying more or less parallel with the epithelial surface in man although in animals they tend to have a more perpendicular axis with respect to the mucosal surface. The ducts of the glands take a perpendicular course and open on the epithelial surface. They are lined with flattened cells. Beneath the epithelial layer, in man, these ducts are frequently enlarged into cystic expansions, also lined with flattened cells. The glandular cells are cuboidal to low columnar, with broad bases giving them a truncated pyramidal form. They contain minute secretory granules. The secretion of Bowman's glands is a serous fluid which bathes the surface of the olfactory epithelium and provides a solvent for the odor carrying substances. It must also serve by its flow to wash away these substances so fresh odoriferous material can exercise its effect. Bundles of olfactory fibers subdivide into numerous smaller twigs that fray out toward the base of the olfactory epithelial layer, their fibers becoming continuous with the olfactory sensory cells. The deeper zone of the tunica propria, which is continuous with the periosteum of the adjacent bones, contains larger blood vessels and a rich lymphatic capillary plexus. Injection masses introduced into the subarachnoid space of the brain cavity

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have been observed to enter the lymphatic plexus of the olfactory mucosa and the sheaths of the olfactory nerve fibers.

In lower vertebrates and in mammals a sac-like formation of epithelium and accessory structures similar to the olfactory membrane constitutes the vomeronasal organ of Jacobson. It is present in the human fetus but regresses in later stages and ordinarily has completely disappeared by the adult stage in man. In many mammals it is large and no doubt serves a function related to that of smell. Apparently it receives stimuli from the mouth cavity by way of the incisive (Stenson's) canals which open into the nasal cavity on each side near the orifice of Jacobson's organ. In man the two incisive canals from the floor of the nasal fossa often join before reaching the incisive foramen, forming a Y-shaped canal, but frequently they are independent as in animals. They are lined with remnants of mucous membrane, connecting that of the nose with the membrane of the roof of the mouth, and are usually closed in adult man but sometimes they are patent as in animals.

The nerves of the olfactory membrane are usually described as the olfactory fila and twigs from the trigeminus. The olfactory nerves and their cells are well known and require no description. The cells are derived from the olfactory placode of the early embryo. They remain in the epithelial layer, as a rule, although Pearson[1, 2] has described some as migrating inward. Their terminal processes, which reach the epithelial surface, are the receptors for smell. Their central processes enter the olfactory bulb. In mammals a vomeronasal nerve is also present. It arises from the same types of neuroepithelial cells as the olfactory nerve. Its fibers enter the accessory olfactory bulb. The vomeronasal nerve and the accessory olfactory bulb are present in the human fetus but regress as Jacobson's organ disappears.

Locy,[3] in 1905, described a ganglionated nerve in Selachians that terminates among the extensive olfactory folds of these fishes, distally, and centrally in the septal portion of the forebrain. Because this part of the brain is morphologically its rostral end, Locy applied the designation *nervus terminalis* to the newly recognized nerve. A similarly located nerve and ganglion had been observed by Fritsch[4] in 1878, and Pinkus,[5] in 1894, had described the connection of the same nerve with the forebrain in the dipnoan fish, Protopterus. De Vries[6] observed the ganglion of the nervus terminalis in the

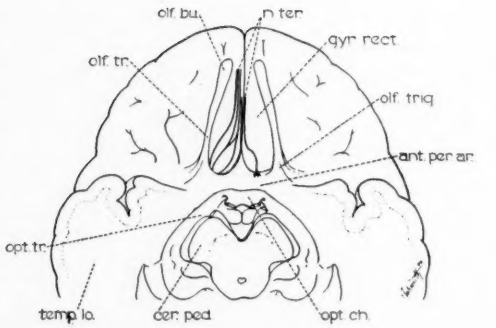

Fig. 1.—Basal surface of human brain into which has been traced J. B. Johnston's (1914) drawings of the nervus terminalis from two human brains. The nervus terminalis on the right hand side of the figure is a mirror image of Johnston's drawing from one brain; that on the left hand side is shown as drawn by him. ant. per. ar., anterior perforated area; cer. ped., cerebral peduncle; gyr. rect., gyrus rectus; n. ter., nervus terminalis; olf. bu., olfactory bulb; olf. tr., olfactory tract; olf. trig., olfactory trigone; opt. ch., optic chiasma; opt. tr., optic tract; temp. lo., temporal lobe.

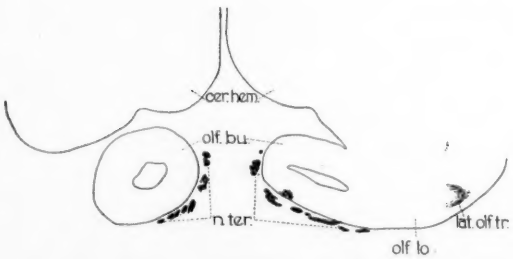

Fig. 2.—Transverse section through olfactory bulbs and anterior part of the cerebral hemispheres showing the roots of the nervus terminalis in a human embryo of 65 mm. C. R. length. Pyridine silver stain. A. A. Pearson Series No. 80. X 20. cer. hem., cerebral hemisphere; lat. olf. tr., lateral olfactory tract; n. ter., nervus terminalis; olf. bu., olfactory bulb, olf. lo., olfactory lobe.

human embryo in 1905. The nerve has since been found in all classes of vertebrates except the cyclostomes. It was discovered in the human adult by Johnston[7] in 1914, and independently the same year by Brookover.[8] These two authors described its intracranial course in man, reaching substantially the same conclusions, namely, that it lies on the surface of the gyrus rectus and enters the brain substance in the region of the medial olfactory stria (Fig. 1). McCotter[9] has described the intracranial and the septal plexuses of the nerve in the human fetus, noting the continuity of the plexuses through the cribriform plate. In our sections of human fetuses the roots of the terminal nerve and its intracranial plexus are prominent from the cribriform plate to the entrance of the roots into the septal region of the brain and the olfactory lobe (Figs. 2, 3, 14-16, 18), especially along the medial surface of the olfactory bulb.

In 1917 Brookover[10] described the peripheral distribution of the nerve in pyridine silver preparations of the nasal septa of two stillborn human infants at about full term. He found an amazingly large anastomosing plexus of nerve fibers, among which were intermingled numerous clusters of ganglion cells. The number of peripheral fibers observed by Brookover greatly exceeded the central root fibers, and he counted more than 1500 ganglion cells in the plexus of the nasal septum. These facts, together with the close proximity of many of the fibers to the blood vessels of the vascular plexus of the nasal septum strongly suggested that the nerve is of autonomic type. Huber and Guild[11] had found a similar extensive peripheral distribution of the nerve in the rabbit and had also pointed to its probable autonomic nature. In 1918 the present writer[12] published the results of a study of the nerve in a number of mammals and in man. This investigation pointed to the presence of vasomotor and of sensory fibers, but with no clear indications of the peripheral or the central connections of either type.

It has been shown in mammalian and human embryos that the ganglion of the nervus terminalis is derived from the olfactory placode (Stewart;[13] Pearson[1]), while Simonetta[14] recognized two parts of the so-called olfactory ganglion (which is the ganglion of the nervus terminalis) namely, a medial part which includes cells resembling neuroblasts, and a lateral part, including small cells. The further development of the terminalis ganglion in the human embryo has been described by Pearson.[15] Simonetta concluded from his

studies that the nervus terminalis in mammals cannot be a sympathetic nerve but has characteristics closely resembling those of a sensory nerve. Pearson[1, 2, 15] has made detailed studies of the development of the vomeronasal nerve, the olfactory nerve and the nervus terminalis. He states with reference to the latter: "The concensus is that the nervus terminalis is functional in mammals and that there are sensory and autonomic components."[15] With reference to the central roots Pearson described them as entering the septal region of the brain and the ventromedial surface of the forebrain just caudal to the attachment of the olfactory bulb.

Peripheral course of the nervus terminalis. The distribution of the terminalis plexus on the nasal septum has been described by McCotter,[9] Brookover,[10] and Pearson[15] in human fetuses; and by Huber and Guild,[11] and Larsell[12] in other mammals. The present investigation, based on serial sections of the entire head, save the lower jaw, of eight young mice stained by the Golgi method; on Golgi sections of the nasal septum and adjacent regions of the kitten; and numerous series of mouse and human embryos stained by the Bodian, pyridine silver and other silver methods all confirm descriptions already made by other observers. It must be recognized that both in the peripheral plexus and intracranially there is the possibility of fibers from other sources, as described below. The fibers of the nervus terminalis, however, appear to stain differentially with the pyridine silver method (Huber and Guild,[11] Brookover,[10] Larsell,[12] Pearson[15]) and with metheylene blue (Larsell[12]). Until experimental evidence clearly gainsays the histological indications the peripheral plexus and its ganglia may be considered as belonging entirely to the nervus terminalis, as described by the investigators cited.

In our silver stained material of human fetuses the main terminalis bundles break up into numerous smaller strands that branch and anastomose with each other in such a manner as to form an elongated plexus (Fig. 3). This lies medial to the olfactory bulb among the olfactory fibers in part, and continues forward on the cranial floor to the foramina of the cribriform plate. Clusters of ganglion cells occur throughout the extent of the plexus within the cranial cavity. Many such cells can be seen among the interlacing bundles of olfactory fibers that cover much of the olfactory bulb before entering it. These obviously represent cells of the embryonic ganglion terminale. Many of them are bipolar but multipolar ele-

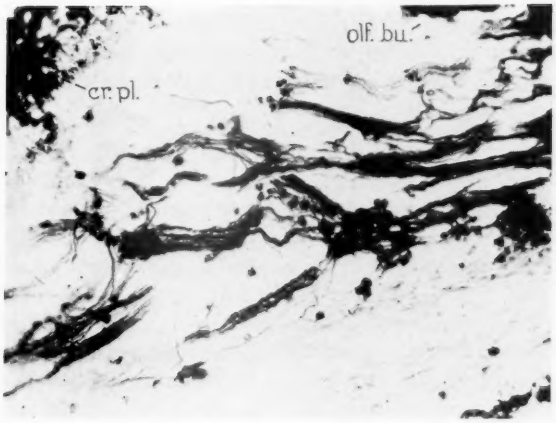

Fig. 3.—Photomicrograph of intracranial plexus of nervus terminalis on floor of cranial cavity rostromedial to olfactory bulb. Embryo of 80 mm. C. R. length. Pyridine silver stain. A. A. Pearson Series No. 83. cr. pl., cribriform plate; olf. bu., olfactory bulb.

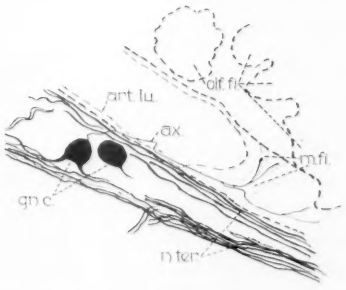

Fig. 4.—Portion of intracranial plexus immediately rostral to lower part of olfactory bulb of human embryo 80 mm. C. R. in length, showing two ganglion cells, with axon process of one ending on muscle fibers in wall of artery. Camera lucida drawing X 580. Pyridine silver stain. A. A. Pearson Series No. 83, art. lu., lumen of artery; ax., axon; gn. c., ganglion cells; m. fi., muscle fibers in arterial wall; n. ter., nervus terminalis; olf. fi., bundles of olfactory fibers rostral to olfactory lobe.

ments also occur. Axons pass from some of the latter to neighboring intracranial blood vessels (Fig. 4). The olfactory cribrosa is intertwined with a very rich plexus of capillaries supplied by these vessels. Some of the multipolar nerve cells in the nerve plexus on the cranial floor appear to send axons to glands of Bowman just beneath the cribriform plate.

The bundles of the intracranial plexus pass through the foramina of the cribriform plate with the bundles of olfactory fibers and spread on the nasal septum and to the olfactory mucosa of the lateral wall of the nasal cavity. Whether or not all of the intracranial cells and fibers belong to the nervus terminalis we have been unable to determine, but they correspond to the intracranial plexus of terminalis fibers as described by previous investigators. The possibility remains of sympathetic fibers from the superior cervical ganglion by way of the internal carotid plexus and its branches on the cerebral vessels; such possible fibers can only be ruled out by experimental methods. Autonomic fibers may reach the terminalis plexus of the septal region of the nasal cavity, and possibly also the intracranial plexus, by way of branches of the trigeminal nerve, although the latter appears unlikely.

The present account of the peripheral plexus will be limited to a more complete description of the ganglion cells, as shown by the Golgi method, and to the motor and sensory endings of the terminalis fibers in the olfactory membrane.

Innervation of septal vascular plexus. Beneath and between the glands of Bowman there is a rich vascular plexus already mentioned. In young mice treated by the Golgi method this shows as a capillary plexus (Fig. 5) the endothelium of which frequently is stained brown. Scattered along the small blood vessels are found numerous multipolar cells having the appearance and staining qualities of ganglion cells (Figs. 5 and 6). Axon-like processes from these cells pass to the small blood vessels. Larger vessels are present deeper in the olfactory membrane. These have accompanying nerve fibers that occasionally branch into the vascular walls. In young kittens and in the adult dog material studied the larger vessels also show nerve fibers.

The relation of the nervus terminalis fibers to the presumptive nerve cells scattered in the capillary plexus in the Golgi sections of young mice has baffled analysis. These cells stain black by the

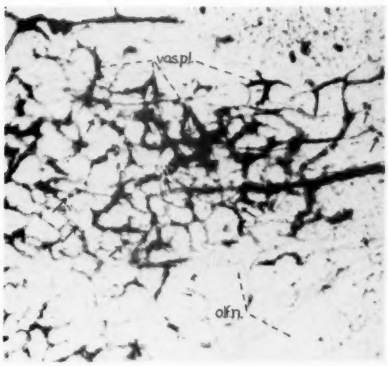

Fig. 5.—Photomicrograph of a portion of the vascular plexus in upper part of nasal septum of 1 day old mouse showing cells and some of the fibers of the apparent nervous plexus. Golgi stain. Medium magnification. Arrows point to apparent nerve cells, many more of which are hidden by the blood vessels or are out of focus; olf. n., bundles of olfactory nerve fibers; vas. pl., vascular plexus.

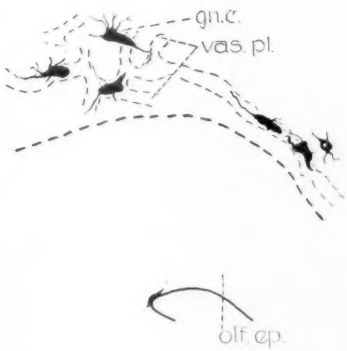

Fig. 6.—Drawing of a small portion of the vascular plexus of nasal septum and some of the ganglion cells in 1 day old mouse. Camera lucida drawing, X 246. Golgi stain. gn. c., ganglion cell; olf ep., olfactory epithelium; vas. pl., vascular plexus.

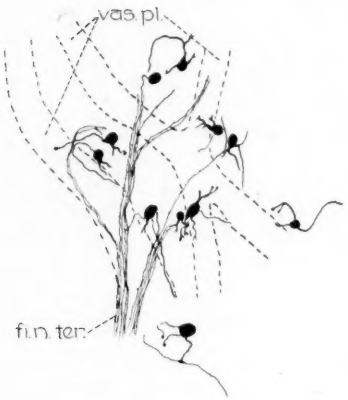

Fig. 7.—Nervus terminalis plexus and ganglion cells in deeper portion of olfactory membrane of 4 day old mouse. Camera lucids drawing, X 246. Golgi stain. fi. n. ter., fibers of nervus terminalis; vas. pl., vascular plexus (larger vessels).

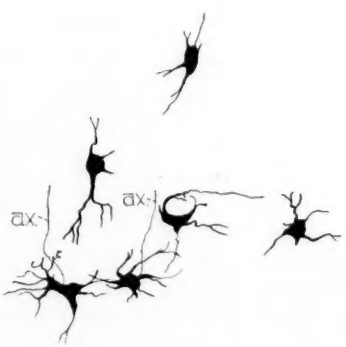

Fig. 8.—Ganglion cells of nervus terminalis in nasal septum of 9 day old kitten. Camera lucida drawing, X 246. Golgi stain. ax., axon.

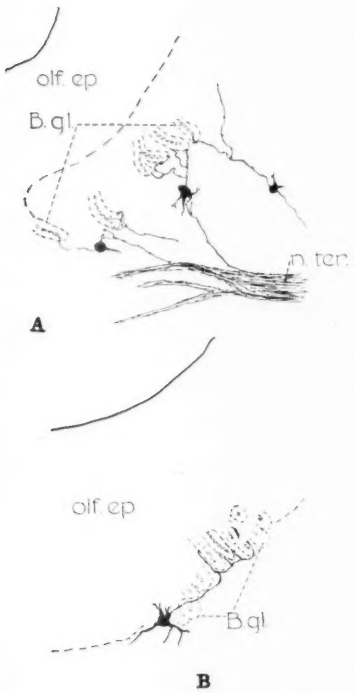

Fig. 9.—Ganglion cells of nervus terminalis plexus with axons to Bowman's glands in 1 day old mouse. *A*, From portion of olfactory membrane immediately beneath the cribriform plate, cut vertically. *B*, From portion of olfactory membrane immediately beneath the cribriform plate, cut obliquely. Camera lucida drawing, X 274. B. gl., Bowman's glands; n. ter., nervus terminalis; olf. ep., olfactory epithelium.

Golgi method and many show slender axon-like processes that apparently pass to the walls of the blood vessels. Most of them lie close to the vessels or in contact with them, but occasional cells are located between loops of the capillaries. In addition to the axon-like processes, they possess thicker branching prolongations that appear to anastomose with similar processes of neighboring cells. In general the cells under consideration resemble the small cells described by Cajal[16] in the villi of the intestine. The relations of these enteric cells to the submucosal plexus of the intestine are obscure. One

must recognize in the plexus among the capillaries of the olfactory membrane the possibility of a local reflex vasomotor mechanism such as is believed by many to be represented by the small cells of Cajal in the digestive tract.

Deeper in the olfactory mucosa of the mice (Fig. 7) and in Golgi sections of the olfactory membrane of kittens (Fig. 8) undoubted multipolar ganglion cells of autonomic type are present. In the young mice these are small and have short dendritic processes. The axons enter the terminalis plexus or extend to the larger blood vessels beneath the periglandular plexus. Similar but better developed ganglion cells were found in the kitten. In the olfactory membrane of the adult dog, stained by a modification of the Cajal method, ganglion cells were observed in close clusters but their processes could not be followed for any distance.

Actual terminations of nerve endings on the muscle fibers of the tunica media of the larger blood vessels has not been observed in our Golgi material of mouse or kitten, but nerve fibers terminating in the vascular walls have been demonstrated in the olfactory membrane of the dog (Fig. 10, 11). These are believed to be derived from terminalis ganglion cells.

Innervation of olfactory glands. Bowman's glands are richly innervated (Figs. 9-11). In young mice, in which these glands are small, fibers from ganglion cells frequently can be seen extending to the alveoli. Fibers reach the ganglion cells from bundles of terminalis fibers or from olfactory bundles in which the terminalis fibers are mingled. In older material of dog and cat actual continuity of nerve fibers between ganglion cells and glands was not observed, but the fibers illustrated in figures 10 and 11 run with the olfactory bundles and have the appearance of nervus terminalis fibers. In Golgi sections of young mice the axons of ganglion cells lying close to the glands can be seen to branch repeatedly and to reach the gland cells as short terminal twigs ending in small knobs which apparently lie against the cell surfaces (Fig. 9). Glands of Bowman and ganglion cells are also present in the membrane of Jacobson's organ in the mouse and cat material. The relations appear similar to those in the olfactory membrane proper.

Sensory endings. In addition to the olfactory cells there are fine branching fibers that terminate in the deeper portion of the olfactory epithelium, ending between the columnar supporting cells (Fig. 12).

They usually do not appear to extend more than about half way through the thickness of the epithelium but some fibers continue toward the surface. The fibers branch chiefly in the basal portion of the epithelium. The number of twigs that can be distinguished as branches of a given fiber is limited, although in many portions of the numerous sections examined they appear abundant. In such regions, however, the olfactory cells also are usually impregnated, and it is difficult to distinguish intertwining cut axons of these cells from the fibers in question in the thick Golgi sections except by most careful manipulation of the microscope.

The free terminations illustrated in figure 12 resemble endings found by Simonetta[14] in the vomeronasal organ of the lamb, and by Lenhossék in the vomeronasal organ of the rabbit, both by the Golgi method. Simonetta considers the terminations which he observed as belonging to the nervus terminalis. Kolmer[18] illustrates similar endings, diagrammatically, in the olfactory epithelium but regards them as terminals of trigeminal fibers. The endings illustrated in figure 9 were found in the portion of the olfactory membrane of the mouse immediately beneath the cribriform plate, and their fibers pass through the plate into the cranial cavity. Similar endings are numerous in other portions of the olfactory epithelium. Terminations of trigeminal fibers are abundant in the respiratory mucosa but, as shown in our Golgi sections, these consist of larger and more freely branching terminal fibers and have a much greater spread in the epithelium. They differ in appearance in other respects also. The type of ending illustrated in figure 9 appears to be confined to the olfactory epithelium, including that of Jacobson's organ. Their fibers enter the bundles of the nervus terminalis and in many cases can be followed to the cribriform plate. They are regarded as afferent endings of this nerve.

A few bipolar cells were found in the Golgi sections of young mice below the cribriform plate (Fig. 12B). On the cranial side of this bone large numbers of such cells can be identified along the course of the nervus terminalis fibers wherever the latter are sufficiently segregated from olfactory fibers to be easily distinguished (Fig. 13). Among the intertwining plexuses of olfactory fibers immediately rostral and medial to the olfactory bulb similar cells are also encountered. Apparently these are cells of afferent terminalis fibers that form part of intracranial plexus of the nerve.

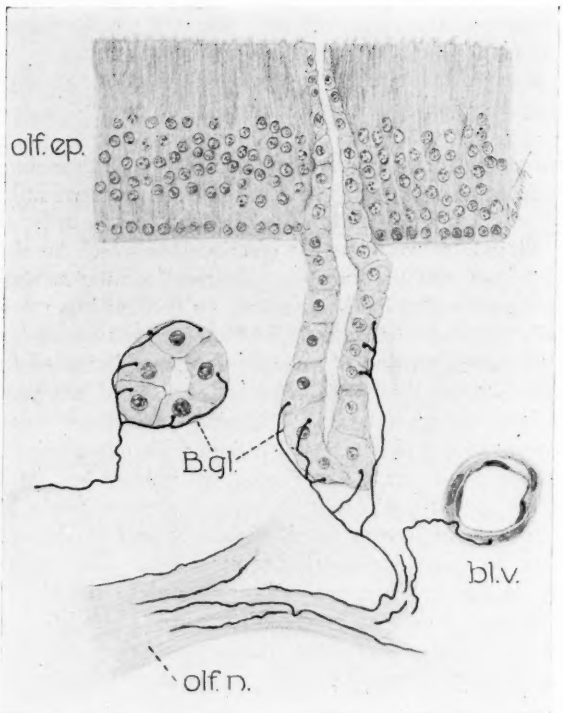

Fig. 10.—Superficial portion of olfactory membrane of dog showing nerve endings in Bowman's glands and in a small blood vessel. Only two of Bowman's glands were included in the figure. Camera lucida drawing, X 346. Modified Cajal stain. B. gl., Bowman's gland; bl. v., blood vessel; olf. ep., olfactory epithelium; olf. n., olfactory nerve.

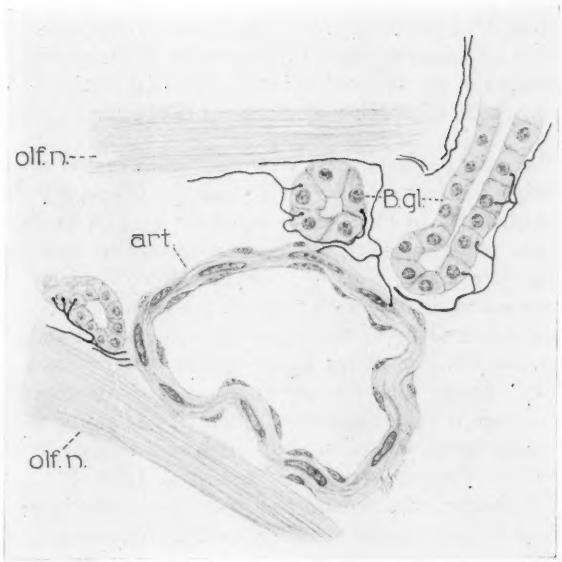

Fig. 11.—Deeper portion of olfactory membrane of dog showing nerve endings in Bowman's glands and in tunica media of a larger blood vessel. Camera lucidà drawing, X 346. Modified Cajal stain. art., artery; B. gl., Bowman's gland; olf. n., olfactory nerve.

Central connections. The roots of the nervus terminalis, in the form of several bundles, course posteriorly along the olfactory bulb and then dip into the brain substance. In the younger human embryos, as described and illustrated by Pearson,[15] they pass into the septal area of the brain, with a bundle turning downward toward the hypothalamus. These fascicles are shown in figure 14, from an embryo 35 mm. in length. In an embryo of 45 mm. several rootlets enter the olfactory lobe ventrolateral to the septal region, and in a 65 mm. embryo, owing apparently to the growth of the olfactory lobe, the bundles have been carried farther laterally within the lobe, four or more fascicles appearing in sections, as illustrated in figure 15. These bundles can be followed through the serial sections to the several entering roots, of which they represent the internal continuations.

All the roots in the embryo enter the brain rostral to the sulcus limitans trigoni olfactorii of Hochstetter.[19] Johnston's[7] figures of the terminalis roots in the human adult show them in this relation in one brain, while in another he illustrates rootlets penetrating the medial olfactory tract (Fig. 1). Brookover's[8] figure of the adult human nervus terminalis shows most of the external roots extending to the rostral boundary of the medial olfactory tract only, but one rootlet on the left side of his figure appears to enter the trigonum olfactorium. In my earlier study of the nerve in mammals I found a similar variation. The most dorsal roots pass laterally and caudally to the medial and the lateral septal nuclei, as described by Pearson.[15] In these nuclei they fray out and terminate (Fig. 16). Many of the fibers, however, arch around the sulcus limitans trigoni olfactorii and turn sharply caudally, passing to the precommissural region. They partly encapsulate and then enter an oval group of small cells (Figs. 16, 18, m.is.C?) which lies rostroventral to the anterior commissure. This cell mass occupies approximately the position of a large island of Calleja shown by Herrick[20] in the opossum, by Obenchain[21] in Caenolestes, by Young[22] in the rabbit and by Crosby and Humphrey[23] in the adult human brain. It may, however, correspond to the dorsal septal nucleus described by Loo[24] in the opossum. In the absence of detailed studies of this region in the embryonic human brain I can only offer the nuclei suggested as possibly representing the cell mass in question. In addition to the small cells which constitute the greater part of this nucleus (Fig. 17) it includes a group of paler staining large cells whose nature could not be determined in the preparations available. This group of larger cells also receives

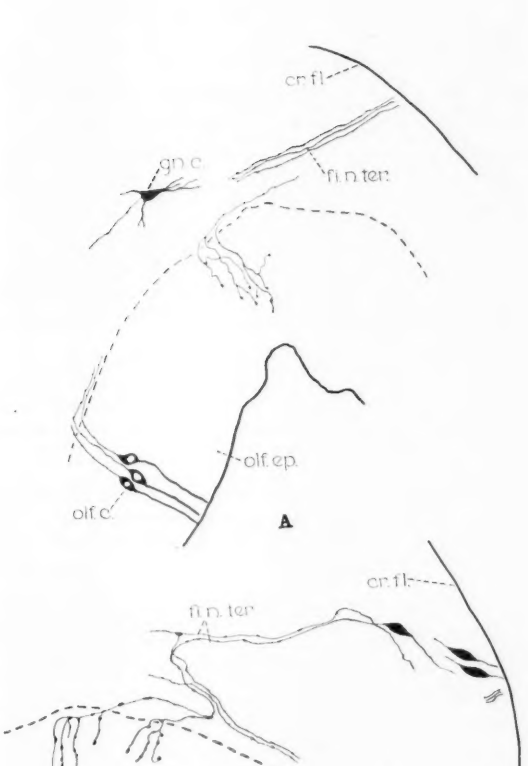

Fig. 12.—Sensory endings of nervus terminalis in olfactory epithelium of 1 day old mouse. *A*, In olfactory epithelium immediately beneath cribriform plate. *B*, Endings in similar position, with nerve fibers attached that show bipolar ganglion cells. Camera lucida drawing, X 205. Golgi stain. cr. fl., cranial floor; fi. n. ter., fibers of nervus terminalis; gn. c., multipolar ganglion cell of nervus terminalis plexus; olf. ep., olfactory epithelium.

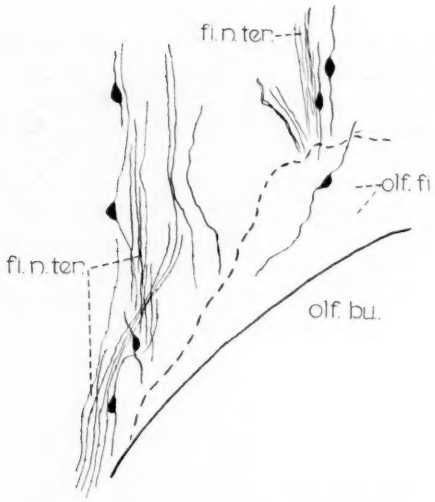

Fig. 13.—Bipolar cells of sensory type in intracranial plexus of nervus terminalis of 4 day old mouse. Camera lucida drawing, X 308. Golgi stain. fi. n. ter., fibers of nervus terminalis; olf. bu., olfactory bulb; olf. fi., zone of olfactory fibers covering olfactory bulb; olf. bu., olfactory bulb.

branching terminal fibers from the same loose bundle that ends, in part, among the small cells. McKibben[25] and Herrick[26] found terminalis fibers in Necturus in the anterior commissure but I have been unable to trace any to this commissure in the human material available. A second diffuse bundle of caudally directed fibers passes downward toward the region of the supraoptic nucleus, these undoubtedly corresponding to the fibers that reach the hypothalamus described by Herrick in amphibians.

The more ventral roots of the nerve in the human fetus enter the olfactory lobe rostral to the sulcus limitans of Hochstetter. Within this part of the brain they form conspicuous bundles (Fig. 15). Fibers from the more medial roots of this group extend laterally, frequently arching toward the entrance point of the next laterally adjacent root. The bundles tend to converge and intermingle at nodal areas of cells. Fine branches appear to terminate among these cell groups, while other fibers spread among the more diffuse cells of this part of the olfactory lobe, which corresponds to the rostral portion

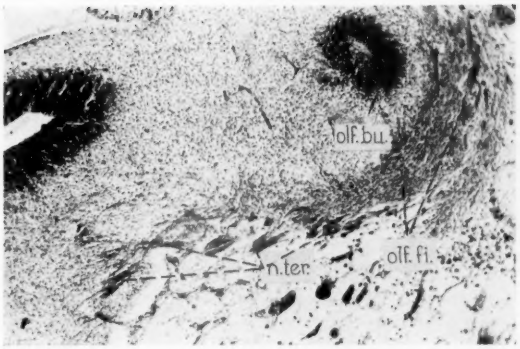

Fig. 14.—Photomicrograph of sagittal section through olfactory bulb and adjacent parts of human embryo of 35 mm. C. R. length, showing roots of nervus terminalis entering the septal area. Silver-gelatine method. A. A. Pearson Series No. 135. olf. bu., olfactory bulb; olf. fi., olfactory fibers; n. ter., roots of nervus terminalis.

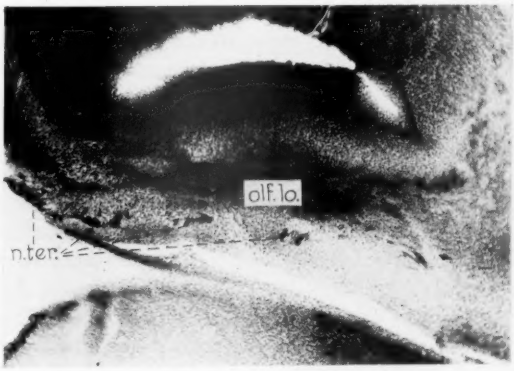

Fig. 15.—Photomicrograph of portion of olfactory lobe of human embryo of 65 mm. C. R. length showing roots of nervus terminalis. Pyridine silver stain. A. A. Pearson Series No. 80. n. ter., roots of nervus terminalis; olf. lo., olfactory lobe.

of the anterior perforated substance of the adult brain. Some of the larger cells of this region give off axon-like processes that pass into the terminalis bundles.

The central terminalis fibers thus reach four regions of the brain, namely, the septal nuclei, the immediate precommissural region (medial island of Calleja?), the olfactory tubercle and the region dorsal and rostral to the supraoptic nucleus.

DISCUSSION

With the demonstration of afferent terminations in the olfactory epithelium, of bipolar ganglion cells connected with its fibers, and of the terminations of its central roots in the septal nuclei, the olfactory tubercle, and the precommissural region of the forebrain, a sensory component of the nervus terminalis appears to be established.

The strong probability is also indicated of a motor component of the nerve reaching Bowman's glands, the blood vessels of part of the nasal mucosa, and the vessels of the olfactory region (at least) of the cranial cavity. While in the porpoise the entire olfactory apparatus, including the organ of Jacobson, is lacking, Johnston[27] has described the nervus terminalis nevertheless as present, as in other mammals and in lower vertebrates. Whether or not a sensory component occurs in the porpoise remains to be ascertained. If it is not present in this mammal the apparent vasomotor component of the nervus terminalis would account for the persistence of the nerve.

If the ganglion cells and their axons to blood vessels and glands are related to the nervus terminalis, as they appear to be, the central root of the nerve must include preganglionic fibers. The central fibers that reach the septal nuclei, the olfactory tubercle, and the precommissural region are undoubtedly sensory by reason of the regions reached as well as of their mode of termination as fine branching processes in these parts of the brain. The fibers extending toward the supraoptic region have not been observed to branch in a similar manner. There are indications that many of them are continuous with cells just anterior to this region. Since the supraoptic nucleus gives rise to fibers to the hypophysis that must be regarded as motor it would not be surprising if the supraoptic region also includes motor cells of preganglionic type to the ganglia of the olfactory region. The evidence, however, is only suggestive and must await more favorable Golgi or other silver stained sections for clear demonstrations.

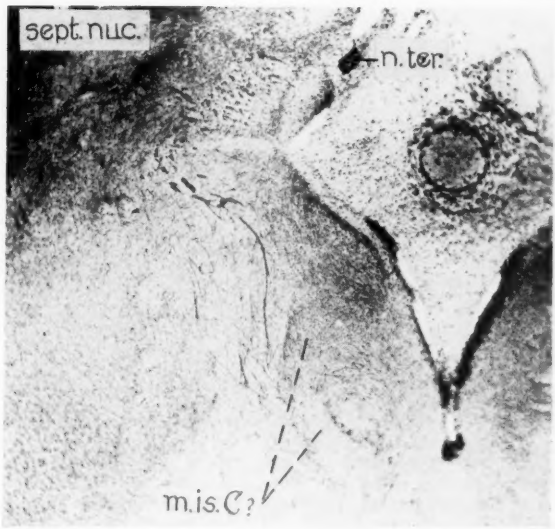

Fig. 16.—Photomicrograph of horizontal section through brain of embryo of 65 mm. C. R. length, showing distribution of fibers of nervus terminalis to septal nuclei and precommissural region. The latter terminate as fine twigs in the oval cell mass tentatively interpreted as the m. is. C?, medial island of Calleja; n. ter., nervus terminalis; sept. nuc., septal nuclei.

The function of the sensory fibers of the nervus terminalis can only be a matter of speculation since there is lack of any observational or experimental evidence of any sort. The type of endings in the epithelium is one of the simplest that has been described for afferent nerve terminations anywhere in the body, even if the branching is considerably more complex than is shown in Fig. 12. The terminations may serve as receptors that are stimulated by the secretion from Bowman's glands which probably is discharged when olfactory stimuli are active. Perhaps impulses produced by flow of this secretion over the olfactory epithelium control and regulate its production and discharge. There is also the possibility that these receptors are stimulated by chemical factors other than those that affect the olfactory cells proper, or those that stimulate trigeminal nerve endings in the respiratory mucosa of the nasal cavity described by Allen.[28] The

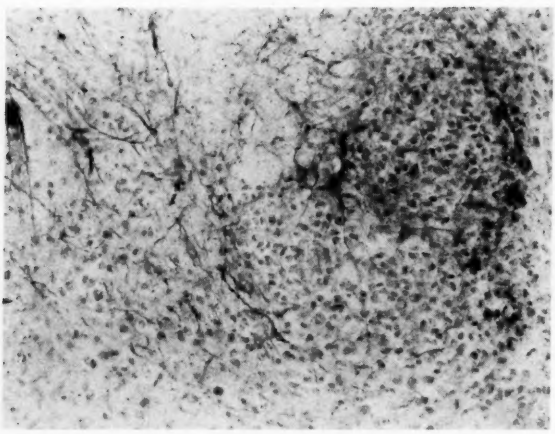

Fig. 17.—Photomicrograph of the medial island of Calleja (?) at higher magnification, showing crowded small cells and the cluster of paler large cells described in text, with nervus terminalis fibers ending among both groups of cells.

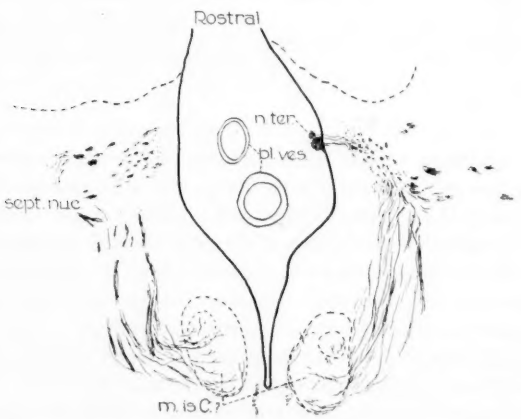

Fig. 18.—Drawing of a section from same series as figure 16, showing on the right hand side the most dorsal root, and the internal distribution of the most dorsal root of the nervus terminalis on both sides. Edinger projection apparatus. X 70. bl. ves., blood vessels; m. is. C?, medial island of Calleja; n. ter., nervus terminalis; sept. nuc., septal nuclei.

fact that many of the root fibers of the terminalis reach the olfactory tubercle, in which secondary olfactory fibers also end, would indicate that they carry impulses differing from those brought by olfactory fibers but which are correlated with the latter. The fibers to the septal nuclei and to the precommissural region likewise probably carry impulses that are correlated with olfactory and other impulses at a reflex response level. It is not clear whether or not any of the fibers to the supraoptic region terminate in the same manner as the others described, i. e., as fine branching processes.

If, as already suggested, the sensory endings of the nervus terminalis serve as receptors that govern the flow of secretion from Bowman's glands they would be accessory to the olfactory apparatus. The nervus apicis described by van Wijhe[29] in Amphioxus, which has no olfactory sacs, is regarded as morphologically homologous to the nervus terminalis. The apical nerve, however, ends in the epithelium of the rostral part of the snout. Its function is unknown but may be chemoreceptor in nature. With the infolding of the olfactory placode to form the olfactory membrane the nervus terminalis of chordates above Amphioxus innervates the epithelium of the membrane and may constitute some modified form of a primitive chemoreceptor.

The nature of the responses to the afferent impulses can only be surmised. The most plausible possibility, so far as reflex responses are concerned, is control of the activity of Bowman's glands. If it may be postulated that the nerve endings in the olfactory epithelium are chemoreceptors stimulated by changes in the fluid secreted by Bowman's glands or by saturation of this fluid by odoriferous substance so that it must be washed away to allow fresh stimuli to exercise their effect, these glands would presumably be set into activity to produce the necessary flow. Since nervus terminalis sensory fibers appear largely to coincide in their distribution with that of the olfactory epithelium, including that of Jacobson's organ, the suggestion of a function accessory to that of the olfactory cells proper seems worthy of consideration. Perhaps the responses provide favorable conditions for the functioning of the special sensory cells of olfaction.

The apparent efferent fibers of the nervus terminalis would seem to constitute the pathway for secretary reflexes to Bowman's glands and for vasomotor reflexes to the blood vessels supplying these

glands. A somewhat similar innervation of the area vasculosa and Shambaugh's gland of the cochlear duct is suggested through the discovery by G. L. Rasmussen[30] of autonomic fibers from the accessory superior olivary nucleus to the cochlea. Presumably these fibers govern the formation of edolymph, while the apparent motor fibers of the periphelar plexus of the nervus terminalis supply the glands and blood vessels of the olfactory membrane, and possibly adjacent blood vessels. The intracranial plexus of the nervus terminalis, in addition, appears to be related to the blood vessels of the olfactory bulb.

SUMMARY AND CONCLUSIONS

1. Afferent nerve endings consisting of simple branched fibers are found in the olfactory epithelium. They differ in complexity and in appearance from endings of the trigeminal nerve in the respiratory mucosa and are connected with fibers of the nervus terminalis.

2. In young mice the afferent fibers of the nervus terminalis are processes of bipolar ganglion cells, some being found in the nasal mucosa just outside the cribriform plate, but the majority occurring along the intracranial plexus of the nervus terminalis and among the meshes of the olfactory bundles outside the olfactory bulb.

3. The roots of the nervus terminalis end in the septal nuclei, the olfactory lobe, the posterior precommissural region, and the anterior portion of the supraoptic region of the brain. Those to the septal nuclei, olfactory lobe, and precommissural region undoubtedly are sensory roots. Those to the supraoptic region may include emergent preganglionic fibers.

4. The cells of the terminalis ganglia of the nasal septum are small, multipolar neurons of autonomic type. Their axons pass to Bowman's glands and, apparently, to blood vessels of the olfactory mucosa.

5. There is evidence that ganglion cells of the intracranial plexus of the nervus terminalis send axons to the blood vessels related to the olfactory bulb.

6. The possible functional significance of the nervus terminalis has been discussed.

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REFERENCES

1. Pearson, A. A.: The Development of the Olfactory Nerve in Man, J. Comp. Neurol. 75:39-66, 1941.

2. Pearson, A. A.: The Development of the Olfactory Nerve, the Nervus Terminalis, and the Vomeronasal Nerve in Man, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 51:317-333, 1942.

3. Locy, W. A.: On a Newly Recognized Nerve Connected with the Forebrain of Selachians, Anat. Anz. 26:33-63, 1905.

4. Fritsch, G.: Untersuchungen über den feineren Bau des Fischgehirns, Berlin, 1878.

5. Pinkus, F.: Die Hirnnerven des Protopterus annectens, Morph. Arb. 4: 275-346, 1895.

6. De Vries, E.: Note on the Ganglion Vomeronasale, K. Akad. van Wetenschappen te Amsterdam 7:704, 1905.

7. Johnston, J. B.: The Nervus Terminalis in Man and Mammals, Anat. Rec. 8:185-198, 1914.

8. Brookover, C.: The Nervus Terminalis in Adult Man, J. Comp. Neurol. 24:131-135, 1914.

9. McCotter, R. E.: A Note on the Course and Distribution of the Nervus Terminalis in Man, Anat. Rec. 9:243-246, 1915.

10. Brookover, C.: The Peripheral Distribution of the Nervus Terminalis in an Infant, J. Comp. Neurol. 28:349-360, 1917.

11. Huber, G. C., and Guild, S. R.: Observations on the Peripheral Distribution of the Nervus Terminalis in Mammalia, Anat. Rec. 7:253-272, 1913.

12. Larsell, O.: Studies on the Nervus Terminalis: Mammals, J. Comp. Neurol. 30:3-68, 1918.

13. Stewart, F. W.: On the Origin of the Ganglion Cells of the Nervus Terminalis in the Albino Rat, J. Comp. Neurol. 32:99-111, 1920.

14. Simonetta, B.: Origine e Sviluppo del Nervo Terminale nei Mammiferi. Sua Funzione e suoi Rapporti con l' Organo di Jacobson, Z. Anat. Entw.-gesch. 97:425-463, 1932.

15. Pearson, A. A.: The Development of the Nervus Terminalis in Man, J. Comp. Neurol. 75:39-66, 1941.

16. Ramón y Cajal, S.: Los Ganglios y Plexos Nerviosos del Intestino, Madrid, 1893.

17. Von Lenhossék, M.: Die Nervenursprünge und Endigungen in Jacobson'schen Organ des Kaninchens, Anat. Anz. 7:628-635, 1892.

18. Kolmer, W.: Geruchsorgan, in Möllendorff, Handbuch der mikroskopische Anatomie. 3:192, 1927.

19. Hochstetter, F.: Beitrag zur Entwicklungsgeschichte des menschlichen Gehirns I. Teil. Franz Deuticke, Wien und Leipzig, 1919.

20. Herrick, C. Judson: The Nucleus Olfactorius Anterior of the Opossum, J. Comp. Neurol. 37:317-359, 1924.

21. Obenchain, J. B.: The Brains of the South American Marsupials Caenolestes and Orolestes, Pub. Field Mus. Nat. Hist., Zool. Series 24: 175-232, 1925.

22. Young, M. Wha .on: The Nuclear Pattern and Fiber Connections of the Non-Cortical Centers of the Telencephalon of the Rabbit (Lepus cuniculus), J. Comp. Neurol. 65:295-402, 1936.

23. Crosby, Elizabeth C., and Humphrey, Tryphena: Studies of the Vertebrate Telencephalon. II. The Nuclear Pattern of the Anterior Olfactory Nucleus, Tuberculum Olfactorium and the Amygdaloid Complex in Adult Man, J. Comp. Neurol. 74:309-352, 1941.

24. Loo, Y. T.: The Forebrain of the Opossum, Didelphis virginiana, Part II. Histology, J. Comp. Neurol. 52:1-148, 1931.

25. McKibben, P. S.: The Nervus Terminalis in Urodele Amphibia, J. Comp. Neurol. 21:261-309, 1911.

26. Herrick, C. Judson: The Amphibian Forebrain. VI. Necturus, J. Comp. Neurol. 58:1-288, 1933.

27. Johnston, J. B.: Nervus Terminalis in Reptiles and Mammals, J. Comp. Neurol. 23:97-120, 1913.

28. Allen, W. F.: A Comparative Study of the Olfactory and Trigeminal Reflexes Elicited by Various Vapors in Different Mammals, J. Wash. Acad. Sciences 26:466-473, 1936.

29. Van Wijhe, J. W.: On the Nervus Terminalis from Man to Amphioxus, K. Akad. van Wetenschappen to Amsterdam, Sect. of Sciences, vol. 21, 1919.

30. Rasmussen, G. L.: The Olivary Peduncle and Other Fiber Projections of the Superior Olivary Complex, J. Comp. Neurol. 84:141-220, 1946.

XXXVIII

THE PATHOLOGY OF ACUTE SUPPURATIVE FRONTAL SINUSITIS

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Anatomically, the frontal sinus is unique among the paranasal sinuses in that the cavity itself may have many variations in size and shape. There may be certain structural factors present which affect adequate drainage and aeration, such as septa from the anterior or posterior walls which more or less completely subdivide the sinus cavity, or aberrant fronto-ethmoidal cells which cause a narrowing of the nasofrontal duct. Likewise, the nasofrontal duct itself offers a greater hazard to drainage compared to the simple ostiums of the other paranasal sinuses. However, this may afford greater protection to the frontal sinus and may, to some extent, explain the infrequency of infections of this sinus in comparison with those of the other sinuses.

The essentials for adequate drainage of the frontal sinus are a patent nasofrontal duct and a normal ciliary activity, not only within the sinus but also in the nasofrontal duct. Another favorable factor is the force of gravity which impels the discharge towards the ostium of the nasofrontal duct.

The frontal sinus is lined with a mucosa on the surface of which is an epithelium of low columnar ciliated type underneath which is a layer of oval basal cells lying on an ill-defined basement membrane in confluence with the tunica propria. The subepithelial areolar connective tissue, or tunica propria, is thinner than that of the maxillary sinus and contains the blood and lymph capillaries, nerve fibers and a sparse scattering of lymphocytes, plasma cells, young connective tissue cells and histiocytes. The glands, few in number,

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are of the serous and mucus secreting types which discharge their secretions to the surface through ducts which penetrate the epithelium. The deeper layer of the tunica propria is continuous with the periosteum to the extent that it may be considered a muco-periosteum.

The periosteum is attached to the bone by fibrils of connective tissue extending into minute canaliculi accompanying small thin-walled capillaries which penetrate into the body of the bone to establish anastamoses with the capillaries of the meninges, the external periosteum, the diploic veins or the marrow spaces respectively. The importance of the diploic veins has been succinctly summarized by Mosher[1] after several years of painstaking histological studies of frontal bones. He states that "The diploic veins of the frontal bone are practically always present. They are absent in only 3 per cent of skulls. They connect with the veins of the mucous membrane of the frontal sinus which in infected membrane are found to be very numerous, and in proportion to the thickness of the frontal bone, very large, often measuring a third of the thickness of the frontal bone. The diploid veins are the transportation system of the infection."

A fundamental concept of acute suppurative frontal sinusitis would be an acute infection of the mucosa accompanied by the formation of pus in the sinus cavity. We cannot properly evaluate this intermediate stage of infection without considering the preliminary stage of engorgement of the mucosa and the subsequent stage of osteomyelitis, since one phase may simulate the clinical picture of the other and give rise to serious problems of differential diagnosis.

During the stage of engorgement the mucosa becomes edematous and infiltrated with bacteria, large numbers of red blood cells, plasma cells, young connective tissue cells, polymorphonuclear leucocytes and histiocytes. The capillaries are dilated and engorged and the glands distended. The mucosa may be thickened to the extent of complete obliteration of the sinus cavity and occlusion of the nasofrontal duct. As the edema subsides the cavity of the sinus is re-established, the fluid and cellular contents of the tunica propria are extruded into it and the suppurative stage results. The course of the pathological picture will then be largely determined by the delicate balance between the degree of patency of the nasofrontal

duct and the viscosity or fluidity of the secretions in the cavity. The activity of the cilia at this stage is also all-important. We recognize the fact that large numbers of epithelial cells with their cilia have become detached and destroyed and that the function of the remaining cilia has been hampered by the edema of the epithelium. The rapidity with which this function is regained may be the deciding factor between a receding and a fulminating infection.

During the acute suppurative stage the tunica propria is invaded principally by large numbers of polymorphonuclear leucocytes occurring individually, in clumps about the capillaries and glands, and within the lymphatics, also by numerous other phagocytic cells, histiocytes, endothelial cells and connective tissue cells. The glands may be ruptured. There is likewise marked engorgement and a variable amount of thrombosis of the blood vessels.

We must not overlook the fact that the stage of engorgement may recede entirely or progress slowly or rapidly to the stage of suppuration. In a word, the progress of an acute infection of the frontal sinus is absolutely unpredictable.

An acute suppurative frontal sinusitis is as serious as its sequelae. These sequelae are always in the nature of extension of the infection from the mucosa of the sinus through the bone of the intersinus septum, or that of the anterior or posterior walls or the floor, or into the marrow spaces of the frontal bone itself. Extension of infection through bone does not necessarily imply a tract of necrosis or osteomyelitis.

Several years ago we[2] observed that the subepithelial spread of infection was an important factor in the extension of infection to the middle ear and mastoid as well as to the middle fossa and perisinus space. In like manner the subepithelial tissue of the frontal sinus mucosa accompanies the capillaries through the bony canaliculi of the anterior and posterior walls of the sinus, carrying the perivascular lymphatics to the external periosteum and the venous plexuses of the dura, which is essentially the internal periosteum. Bacteria may be carried through these channels, either free or within phagocytic cells and become established as foci of infection in the respective terminal areas. Thus a subperiosteal or subdural abscess is a logical sequel.

The thrombosis of the venules and the blocking of the lymphatics in the tunica propria are the most significant pathological

changes occurring in the earlier stages of the suppurative period of infection. These vascular changes are forerunners of the retrograde spread of the infection to the intracranial cavity and the narrow spaces of the frontal bone. Mosher[3] and Furstenberg[4] have stressed this point repeatedly.

This retrograde spread of infection is not only accomplished by the actual accumulation of the infected thrombi backward or upward, but also by a reversal of the blood or lymph streams proximal to the zone of obstruction of the vessels.

The type of involvement of the bony walls of the frontal sinus depends upon the walls involved. In the usually compact bone of the intersinus septum, the posterior and the orbital plates, infection results in an osteitis, while in the spongy bone of the anterior wall an osteomyelitis is established. Compact bone may undergo dissolution by necrosis, osteomalacia or rarefaction. In necrosis the vitality of the bone is lost due to a thrombosis of the nutrient arteries, the bone cells disappear, the calcium is absorbed and the integrity of the bone is lost. In osteomalacia there is massive invasion of spontaneously decalcifying bone by young connective tissue cells leading to more or less complete replacement of the bony wall by the invading cells, thus forming a membranous wall continuous with the adjacent periosteum on either side. This change results from a diminution of the blood supply to the bone, thus stimulating the formation of connective tissue. This hypothesis is substantiated by the work of Rohde.[5] Rarefaction may be designated as such when the osteoclasts are seen to be actively destroying the bone. They are found either singly, causing isolated erosions along the edge of the bone, or in large numbers, causing a massive destruction from all sides. The bone retains its deep staining properties and the bone cells remain vital until their lacunae are destroyed by the advancing osteoclasts.[6] Osteoblasts are always present as a normal counterpart of osteoclasts. However, the numbers and activity of the latter in infections of bone greatly outweigh the reparative functions of the former. According to von Gaza[7] paraplastic substances which are released by the dying or dead bone cause the transformation of specific connective tissue cells into osteoblasts, since necrotic and formative bone stand in such close physical and chemical relationship. Perforation of a thin wall may thus occur easily in rapidly advancing rarefaction.

In a fulminating acute suppurative frontal sinusitis the line of distinction is so fine between the infection of the mucosa and extension into the marrow spaces that the two stages might be considered as simultaneous conditions, rather than one as a sequel of the other.

Our present day concept of the pathology of osteomyelitis of the frontal bone has been modified from the classical description of this condition by Mosher and Judd[3] by the use of antibiotics and sulfonamides. Nevertheless, our uncertainty of the extent of this modified pathological picture warrants a repetition of their observations.

They have thus described this condition:

"The gross appearance of infected bone naturally varies considerably with the stage of the disease. In the very early stages of osteomyelitis of the cranial bones, the diploe may show some congestion on cut section but generally no abnormality of the bone structure can be seen. Soon, however, the diploe become markedly hyperemic and contain small drops of pus. The bone appears slightly whiter than normal and its consistency is softened. After seven to ten days, the diploetic spaces are filled with granulation tissue, bathed in pus, which escapes freely from the cut surface of the bone. Thrombosed vessels may or may not be found. The bone itself becomes discolored, at first appearing hemorrhagic but finally becoming dark red in color. As the disease progresses still further, pus may be seen oozing to the surface of the discolored bone through the vascular channels or through small eroded fistular openings. On removing the outer table small sequestra may be found in the diploetic spaces. Finally the infection breaks down the resisting external and internal tables with more or less widespread destruction, marked by collections of pus on the surface of the bone and irregular sequestration of either table.

"Microscopic examination of specimens of bone in the very early stages of the disease show the myeloid tissue to be somewhat edematous, with a congestion of the vessels. There is a moderate infiltration with lymphocytes and polymorphonuclear leucocytes but the bone structure is intact and its margins are smooth. The number of inflammatory cells rapidly increases especially around the blood vessels. Should the infection be blood born, some of the vessels will show septic thrombosis. A few areas will now be found here and there, where the bone cells, or osteocytes, are missing and the lacuna are enlarged by resorption. Such necrotic areas are produced by an obstruction to their blood supply by the inflammatory process. As the infection progresses, the myeloid tissue becomes degenerated and is replaced by granulation tissue and pus. Small chips of necrotic bone are sequestrated and lie free in the granulations. The margins of the bone lining the diploe are eroded and very irregular, giving a moth eaten appearance.

"Osteoclasts appear, surrounding the sequestra, and lining up against the surface of the bone. To all appearances, the activity of these cells consists in further erosion and destruction of the bone. The number of osteoclasts varies considerably in different cases. Occasionally they are difficult to find; again they may lie side by side along the whole margin of the bone.

"Frequently evidences of osseous repair can be found in the areas of destruction. It is not uncommon to find small areas of new formed lamellae, lined by osteoblasts, in specimens that show marked destruction. Bone formation and bone destruction are present at the same time in diseased bone just as in embryonic bone. In the former case, however, the regressive changes predominate until the infected bone has been removed surgically.

"Bone will attempt to raise a barrier against infection just as soft tissues will. Occasionally toward the periphery of the disease, the granulations will be converted into connective tissue. This change however, generally does not become sufficiently completed to arrest the progress of the disease.

"The infiltration and pus formation spreads from the diploe, through the Haversian canals, bringing destruction to the compact bone. The destruction is chiefly by erosion, absorption, and sequestration. The amount of destruction depends upon the severity and duration of the disease."

We feel that in the engorgement and the acute suppurative stages the pathological picture is reversible, especially with the use of adequate and sustained antibiotic and sulfonamide therapy, while in the stage of osteitis and osteomyelitis the condition is less likely to respond to these measures because of the attenuated blood supply due to the thrombosis of the capillaries.

SUMMARY

1. In acute suppurative frontal sinusitis there is a marked edema of the mucosa, an engorgement of the blood capillaries, an infiltration of the tunica propria by red blood cells, large numbers of polymorphonuclear leucocytes, which occur in clumps and about the blood vessels and glands, by histiocytes, endothelial cells and connective tissue cells. There is also a thrombosis of many of the venous channels.

2. The edema may cause obliteration of the sinus cavity and obstruction of the nasofrontal duct. The favorable or unfavorable progress of the clinical condition may be in direct proportion to the timely subsidence of these pathological changes.

3. Involvement of the bony walls of the sinus may occur in the form of a periostitis, an osteitis or an osteomyelitis.

4. The usual pathological picture is undoubtedly modified by the administration of antibiotics and sulfonamides.

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REFERENCES

1. Mosher, H. P.: My Milestones, Laryngoscope 56:639-652 (Nov.) and 736-761 (Dec.) 1946.

2. McMahon, Bernard J.: The Relation of Mastoid Structure to Extension of Mastoid Infection, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 40:53 (Mar.) 1931.

3. Mosher, H. P., and Judd, D. K.: Tr. Amer. Laryng., Rhin. and Otol. Soc., 1933, pp. 328 and 330-333.

4. Furstenberg, A. C.: Tr. Amer. Laryng., Rhin. and Otol. Soc., 1931.

5. Rohde, Carl: Ueber den Ablauf der Regenerations—vorgänge am Röhrenknochen beierhaltener und geschadigter Defässversorgung, zugleich ein Beitrag uber Herkunft und Entstehungsbedingungen des Bindergewebes nach Knochenverletzungen, Arch. f. klin. Chir. 123:530, 1923.

6. McMahon, Bernard J.: Pathology of Spheno-Ethmoidal Sinusitis, Arch. Otolaryng. 4:310-333 (Oct.) 1926.

7. Gasa, von: Die Bedeutung der Gewebszerfallstoffe (Autolysate) für das regenerative Geschehen, Arch. f. klin. Chir. 121:378, 1922.

XXXIX

TREATMENT OF ACUTE FRONTAL SINUSITIS

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In considering the problem of treatment of acute frontal sinusitis it is quite obvious that there is no single procedure which can be followed in all types of cases. Therefore, it is important to evaluate the patient's problems on an individual basis. This entails a careful history and special consideration in regard to the degree of pathology from which the patient is suffering and an accurate study of any anatomical abnormality which the patient may present. This includes bacteriological investigation and also an accurate roentgenological evaluation.

Infection of the frontal sinus may be merely a very slight inflammatory process involving the mucosa of the sinus with partial obstruction of the nasofrontal duct. The patient usually has some elevation of temperature, there is pronounced pain and tenderness and headache which reaches its peak in the forenoon, and the sinus discharges purulent secretion through its normal exit. Such a case does not call for early surgical intervention, in fact the most conservative treatment is indicated. Rest in bed and use of chemotherapy or appropriate antibiotics, codeine and aspirin to control the pain, a shrinking solution applied to the infundibulum to promote drainage, and the application of moderate heat externally are usually sufficient. This treatment is especially applicable to the patient who has never had an acute infection previously. The problem in such cases, however, includes measures to prevent reinfection. It is extremely important to eradicate the organism responsible for the infection. As has been emphasized by Davidson,[1, 10] a careful analysis of the bacteria involved, together with a determination of their sensitivity to the various antibiotic preparations is important in order to ascertain the dosage required for their eradication. It is my opinion that one of the causes for reinfection has been a failure heretofore to recognize the persistence of pathological bacteria within the sinus after the patient has been discharged as clinically cured. In

such cases, reinfection may ensue when the balance between the patient's immunity to that organism and the virulence of the strain has been upset in favor of the bacteria.

In some cases there are structural abnormalities such as a blocking of the infundibulum by crowding of the middle turbinate against the lateral wall, poor ventilation of the nares due to significant deviations of the nasal septum, or encroachment of a fronto-ethmoidal cell which partially obstructs the nasofrontal duct. In such cases it is advisable to perform a sub-mucous resection or to infract or amputate the anterior tip of the middle turbinate depending on the type of abnormality with which one is dealing. The problem of the fronto-ethmoidal cell does not usually come up for surgical consideration unless it is necessary to undertake an extensive operation on the ethmoid or frontal sinus itself.

In the type of case which I have just described, that of early acute and isolated attacks of frontal sinusitis, more extensive surgery is rarely required. However, in case the frontal sinus fails to drain, surgical intervention at an early date may be required. In such a situation an external trephine through the floor of the frontal sinus near its medial portion, insertion of a drain and the early use of chemotherapy and antibiotics will hasten the recovery and go far to prevent serious irreversible damage to the mucosa of the frontal sinus. The drain is left in place until the nasofrontal duct has sufficiently recovered to permit normal drainage into the nose. The drain may then be removed and the wound closed. At times nature attempts to drain the frontal in the same way and we have a perforation through the floor and the formation of an orbital abscess; this should be drained and treated in an exactly similar manner.

It is possible for a patient to have repeated attacks of acute frontal sinusitis such as I have described and recover clinically between attacks but if the frontal sinus suffers sufficient damage so that it progresses to chronic suppuration the picture changes and the surgeon is faced with the decision of advising more radical surgery. It is quite true that many cases of moderate chronic frontal sinusitis, even with an occasional bout of acute infection, may lead a more or less comfortable existence without surgery and therefore many patients refuse operation, and although their sinuses continue to suppurate they are able to carry on activities at a reasonably satisfactory level. However, the reinfection of such cases if fraught with the

most severe dangers such as an extension into the orbit, meninges or blood stream.

In the early stages of an acute exacerbation of chronic sinusitis conservative treatment is still indicated. It is important to localize and limit the inflammatory process and to build up a satisfactory blood level with chemotherapy and antibiotics just as in the acute infection uncomplicated by chronic pathology.

In some cases the subsequent course is quite different. In addition to correction of the intranasal mechanical abnormalities such as a deviated septum, we also have to remove infected tissue from the frontal sinus and to eradicate other areas of infection such as an infected ethmoid, sphenoid or maxillary sinus.

It is generally considered that an operation such as that of Lynch is the one of choice in such a case. The Lynch type, originally outlined by Jensen, requires the removal of the floor of the frontal sinus and a complete ethmoidectomy. There are definite problems connected with such an operation, chief among which is the postoperative obstruction to drainage due to scar tissue in the region of the nasofrontal duct. In such an operation the duct actually is removed if the floor of the sinus and the ethmoidectomy are completed. This allows the periosteum of the orbit to come very close to the interfrontal septum and it is the sad experience of nearly every operator to find that when the surfaces come in contact they become united by scar tissue. Therefore, several modifications of the Lynch operation have been described. The operation as performed by Brown[2] and Walsh[3] preserves the nasofrontal duct with its mucosa. This procedure is based on the assumption that a nasofrontal duct which is relieved of injury due to infection will perform its function more satisfactorily than an artificial opening into the nose. However, there are definitely cases where the nasofrontal duct is so injured that it is doubtful if such conservative operations will succeed. From the very beginning surgeons have attempted to keep the nasofrontal passage clear by artificial means such as rubber drains, gold tubes as advocated by Ingals[4] and later by Goodyear,[5] tissue grafts which have been employed by Negus[6] and Hoople,[7] and acrylic molds by Hoople and New.[8] This author's[9] preference has been tantalum foil which is laid over the orbital periostem as a barrier to the formation of adhesions to the septum. Tantalum has also been used as a tube with satisfactory results by Weille[11] and Brown.

The situation, however, may call for more radical and destructive surgery. In other words, all our attempts to maintain a frontal sinus cavity may be defeated by the presence of such severe infection of the bone that only a complete removal of the front wall, the floor and the posterior wall of the frontal sinus will eradicate the infection. Certainly the retention of osteomyelitic bone, the danger of pocketing off areas of the frontal sinus especially where the sinus is wide and shallow would make the Lynch procedure of doubtful value. Where the infection in one frontal sinus has progressed to the extent that it has eroded the interfrontal septum and secondarily infected the opposite sinus, it will call for a serious consideration of the advisability of obliterating both sinuses. One of the most important don'ts in obliterative surgery is not to leave remnants of mucosa buried in the obliterated sinus. These all too frequently grow and form mucoceles or pyoceles. It is, therefore, my opinion that it is far better to remove all vestiges of mucosa, and in a case involving the opposite sinus as I have described, I would feel on much safer ground if I obliterated both sinuses at the same time. The only objection to the obliterative procedure which has been valid up to the present date has been the resulting deformity. The beautiful cosmetic results following bone implantation for the repair of the frontal defect has completely removed this objection.

While the discovery of antibiotics has vastly changed the picture of upper respiratory infection and has eliminated the necessity for surgery in many cases, nevertheless it is not to be relied upon to the exclusion of surgery. Likewise one should not depend on his surgery exclusively and for this reason I feel that chemotherapy with antibiotic treatment is essential pre-operatively and postoperatively in order to promote more rapid recovery.

And finally, the surgeon should never forget that failure to follow his cases postoperatively may be the undoing of the most brilliant operative technique. Also, the patient must be impressed with the necessity of reporting at regular intervals after the operation and especially whenever there is a fresh upper respiratory infection so that reinfection of the sinuses may be prevented or aborted.

REFERENCES

1. Davison, F. W.: The Penicillin Treatment of Sinus Infection, Pa. Medical Journal, 51:272-275 (Dec.) 1947.

2. Brown, J. M.: Frontal Sinusitis, The Laryngoscope, 6:116-119 (Mar.) 1946.

3. Walsh, T. E.: Experimental Surgery of the Frontal Sinus. The Role of the Ostium and Nasofrontal Duct in Postoperative Healing, The Laryngoscope 53:75-92 (Feb.) 1943.

4. Ingals, E. F.: New Operation and Instruments for Draining the Frontal Sinus, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 14:512 (Sept.) 1905.

5. Goodyear, H. M.: Surgery of the Frontal Sinus, Trans. Amer. Laryng., Rhin. and Otol. Soc., 1947, pp. 64-78.

6. Negus, V. E.: Brit. Med. J., January 25, 1947, pp. 135-136.

7. Hoople, G. D.: An Otologist's Experience in World War II, Trans. Amer. Laryn., Rhin. and Otol. Soc., 1946, pp. 183-193.

8. New, G. B., and Erich, J. B.: An Acrylic Obturator Employed in the Repair of an Obstructed Frontonasal Duct, Archives of Otolaryng., 45:209-214 (Feb.) 1947.

9. Goodale, R. L.: The Use of Tantalum in Radical Frontal Sinus Surgery, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 54:757 (Dec.) 1945.

10. Davison, F. W.: Antibiotics and Sinus Infections, The Laryngoscope, 60: 131-141 (Feb.) 1950.

11. Weille, F. L.: The Problem of Secondary Frontal Sinus Surgery, Transactions of the American Laryngological Association, 1946.

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ACUTE SUPPURATIVE FRONTAL SINUSITIS INTRACRANIAL COMPLICATIONS

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In this period in which the chemotherapeutic and antibiotic agents have combated so successfully the acute suppurative processes in the paranasal sinuses, one might question the need for further discussion of the intracranial complications of these infections. The fact that these complications still occur on occasion in spite of modern medical therapy should serve to stimulate the rhinologist to make a more intensive study of these conditions and to attempt to evaluate his treatment of the acute sinus infections. With few exceptions, each case of intracranial extension of infection from the sinuses bears witness to the fact that the original treatment of the acute sinusitis failed. Nearly all of the acute suppurative processes in the frontal sinuses are amenable to modern medical therapy in the early stages of their development. A review of cases of intracranial complications of these infections indicates that the common causes for failure of treatment of the original infections are:

1. Starting the use of specific drugs too late.

2. Inadequate therapy, either by too small amounts of chemotherapeutic agents, or, timid and intermittent administration of these agents. Too many times as is the case also in otitis media, the patient with an acute frontal sinusitis is given a shot of penicillin, a handful of sulfa pills, and told to return if there is further trouble.

3. Use of non-specific therapy. Most of the acute sinus suppurations respond to penicillin or the sulfonamides. However, one may be lulled into a false sense of security by giving adequate doses of penicillin, for example, to a patient whose acute sinusitis is due to the hemophilus influenzae bacillus. The ideal procedure of a routine bacterial culture in all cases of acute sinus infection may not be feasible in general office practice. If culture is not taken originally, the patient must be kept under observation. The lack of

definite favorable response within 72 hours indicates inadequate therapy and should prompt complete re-evaluation including bacterial culture.

Extension of infection beyond the natural boundaries of the frontal sinuses, and one must include the neighboring ethmoid cells, may occur in either acute or chronic suppurative processes. Since, in the latter case, an acute exacerbation is usually responsible for the spread of infection, consideration must be given to such a condition in this presentation.

In this brief discussion of the intracranial complications of acute suppurative frontal sinusitis, I wish only to consider some of the more important differential diagnostic points referable to these lesions, which in their early stages may present most confusing clinical pictures.

In general, intracranial complications due to an extension of infection beyond the natural boundaries of the involved sinus in the case of *acute* suppuration, occur more frequently in cases of fulminating infection caused by H. influenzae bacillus, those due to the acute exanthematous diseases, and those resulting from swimming or diving. This is particularly true in the adult. In the case of the very young child, extension of such an acute, fulminating infection beyond the bounds of the sinus is much more likely to produce an extracranial collection of pus such as a periorbital abscess. My personal experience would indicate that the *primary* complication resulting from an acute exacerbation of a *chronic suppurative frontal sinusitis* is more likely to be in the form of an osteomyelitis of the skull than in the nature of an intracranial lesion. It is reasonable to assume that in the old chronic suppurative processes, the proliferative bone changes and thrombosis of communicating veins have served to offer some protection against the possibility of continuity of infection through vascular channels.

Most of the intracranial complications of suppurative frontal sinus disease are neuro-surgical problems. As rhinologists, however, we are responsible for the treatment of the acute sinus infections and for the recognition of their complications. By the same token, we must be able to differentiate these complications one from another in order to treat them ourselves if we are adequately prepared, or at least to recognize the need and urgency for medical or surgical treatment at the hands of others who are capable of the task.

Intracranial complications of suppurative processes in the frontal sinuses occur by direct extension of infection through contiguous structures or by way of septic thrombo-phlebitis in communicating veins. Thus, the primary complication may be a localized area of osteomyelitis which may involve both tables of the skull and produce an epidural abscess by this method of direct extension. In acute fulminating cases of frontal sinusitis, particularly in adults, extension of infection may proceed intracranially by means of septic thrombo-plebitis of the numerous communicating veins extending from the sinus submucosa through the intact posterior wall of the sinus to the veins of the dura. Thus, an intracranial infection may complicate an acute frontal sinusitis even before an actual empyema of the sinus has had time to develop.

The intracranial complications of suppurative frontal sinus disease of which the rhinologist must be aware are chiefly:

1. Osteomyelitis of the skull.

   While this is not actually an intracranial complication, it must be included with the group if differential diagnosis is our primary concern.

2. Epidural abscess.
3. Epidural hemorrhage.
4. Diffuse subdural abscess.
5. Thrombosis of the superior longitudinal sinus.
6. Cavernous sinus thrombosis.
7. Leptomeningitis.
8. Diffuse purulent encephalitis.
9. Localized frontal lobe abscess.

This is an impressive list which very obviously can not be considered in any comprehensive manner in the time allotted to this discussion. It must suffice, then, to record a few of the signs and symptoms which are important in making a differential diagnosis when an intracranial extension of a sinus infection is suspected.

First, let us consider the danger signs in suppurative sinusitis; those signs or symptoms indicating a spread of infection beyond the anatomical boundaries of the sinus.

1. External swelling of the forehead, region of the glabella, and eyelids.

2. Generalized headache.

3. Vomiting.

4. Changes in sensorium.

5. Visual disturbances, such as blurring of vision or diplopia.

6. Cranial nerve palsies.

7. Convulsions.

8. Chills and spiking fever.

9. Marked disproportion between signs and symptoms such as changes in sensorium in a patient who has had very recently a fulminating sinusitis from which he seems to be recovering.

10. Signs of acutely increased intracranial pressure (bradycardia, hypertension, and changes in the respiratory cycle).

11. High leukocyte count, that is, above 20,000 or 25,000 in an adult.

None of these signs or symptoms has any place in uncomplicated sinus disease. They indicate disease elsewhere than in the cavity of the sinus which must be located, evaluated, and treated. One's specialized enthusiasm must not make him forget that he is a physician. As such, he must consider the possibility that the patient might have pneumonia, pyelitis, malaria, subacute bacterial endocarditis, migraine, and a host of other general systemic diseases which might account for one or more of the complicating symptoms or signs mentioned above.

OSTEOMYELITIS OF THE SKULL

In conisdering the various complications individually, I shall omit any detailed discussion of this condition. Diffuse, spreading osteomyelitis of the skull, secondary to frontal sinus suppuration, has become a rare lesion since the advent of the newer chemotherapeutic and anitbiotic agents. In an established case of osteomyelitis there is an uncompromising demand for complete excision of the infected bone. Prior to modern medical therapy, radical surgical treatment was required at the onset of the infection on the basis of clinical findings during a period before roentgenographic evidence of the destructive process in the bone was evident. Now, specific

therapy may permit spontaneous healing of the early invasive infection of the diploe and, in an established case, will prevent the devastating spread of the infection through the diploe and over the surface of the dura which was characteristic of this lesion in former years. Consequently, although the treatment is still surgical, the indications for operation have changed remarkably. It is generally advisable to postpone surgical excision of the frontal bone until there is roentgenographic evidence of disease. During this period of waiting, however, closest observation must be maintained to determine any evidence of an intracranial spread of the infection.

EPIDURAL ABSCESS

Epidural abscess is commonly associated with osteomyelitis of the skull. In fact, in every case of established osteomyelitis, one must assume the presence of an epidural abscess. This demands removal of full thickness of the skull in the region of the infection until a wide margin of healthy dura is exposed. Surgical excision must not be limited to the outer table even though all evidence points to limitation of infection to this area. In a frank case of osteomyelitis, the epidural abscess does not present any particular diagnostic problem because it will become apparent and will be drained adequately when full thickness of the calvarium is removed at the time of operation. It is the epidural abscess that may develop early in an acute fulminating sinusitis and appear beneath an intact inner table that produces the difficult diagnostic problem. When this occurs, it is the result of septic thrombo-phlebitis of the communicating vascular channels between the sinus mucosa and the dura. Although there are no pathognomonic indications of this condition, there are likely to be signs, even though equivocal, which should lead one to suspect the possible presence of such a collection of pus:

1. Generalized headache with signs of slight meningeal irritation during the first three or four days of an acute fulminating sinusitis.

2. Continued clinical evidence of undrained pus after trepanation for frontal empyema or after removal of bone for osteomyelitis.

3. Tenderness to percussion beyond the boundaries of the frontal sinus or beyond the pitting edema in an individual who has an osteomyelitis.

Admittedly, these signs may be present in a case of frontal lobe brain abscess; but in the latter, there is commonly a chill at the time of cerebral invasion, the meningeal signs are more prominent, pleocytosis is present, there are likely to be some changes in the sensorium, and commonly some evidence, though perhaps slight, of increased intracranial pressure.

An epidural abscess developing during an acute fulminating frontal sinusitis may become firmly encapsulated and remain latent only to produce a secondary osteomyelitis sometime after the acute sinusitis has resolved completely under medical therapy.

EPIDURAL HEMORRHAGE

To my knowledge, this rare complication of osteomyelitis of the skull is not recorded in the literature. It was called to my attention by Dr. Richard Schneider, of the Department of Neurosurgery, at the University of Michigan. In the near future he will report two cases of this condition. One of these was seen in the University of Michigan Hospital in 1947 and was secondary to a chronic suppurative otitis media and mastoiditis. In this case the condition proceeded rapidly to fatal termination. Because of the original diagnosis of brain abscess, treatment was delayed. Last year, Dr. Schneider informed me regarding another of these cases and has granted me permission to cite it at this time. The patient was a 21 year old male, examined in July of 1949. Five weeks previously, a furuncle on the right nasal ala had been treated and apparently cured by administration of penicillin for four days. Right frontal headache and drowsiness developed three weeks later. At the time of examination there was a small rarefied area in the right frontal bone demonstrated on the x-ray film which suggested an osteomyelitis. There was dilatation of the homolateral pupil. Ventriculagram demonstrated a massive shift of the ventricular system to the left with depression of the frontal horn of the right lateral ventricle. A trephine opening near the osteomyelitic area exposed a massive extradural hemorrhage which had pushed the dura and frontal lobe toward the middle fossa. Cultures of the clot demonstrated a hemolytic staphylococcus aureus. The exact point of erosion of a blood vessel producing the hemorrhage was not demonstrable.

The so-called tentorial pressure cone syndrome is important in the diagnosis of any intracranial hemorrhage. There is dilatation of the homolateral pupil as the hemorrhage increases in size and

pushes the temporal lobe down through the incisura of the tentorium pressing first on the superior colliculus with its underlying third nerve and Edinger-Westphal nuclei. As this herniation increases, pressure is exerted first on one cerebral peduncle and then the other compressing them against the edge of the tentorium and causing the pyramidal tract sign of a Babinski reflex. If the hemorrhage is arterial, the patient may succumb rapidly after these signs develop and before there is any significant alteration in the pulse and blood pressure. If the bleeding is of venous origin, as was probably the case in the patient cited above, the extradural collection of blood forms more slowly and the patient may be able to tolerate the slowly forming tentorial pressure cone for several days.

The rarity of extradural hemorrhage complicating osteomyelitis of the skull is probably due to the fact that there is rather extensive thrombosis of vessels in the osteomyelitic area. Considering the pathological changes present in osteomyelitis of the skull, it would seem likely that if extradural hemorrhage were to occur, that it would be the result of erosion of an artery.

DIFFUSE SUBDURAL ABSCESS

In almost every instance, this is a complication of an acute fulminating sinusitis. Rarely does it develop during an acute exacerbation of a chronic infection. (This condition must be distinguished from the localized subdural abscess occasionally associated with cases of chronic osteomyelitis and which may remain latent for some time.)

The most important diagnostic point is the relatively rapid development of the clinical picture. Usually there are signs of extension to the intracranial cavity within a few days. Perhaps the patient may have a little generalized headache for two or three days during which time he has not been particularly ill. Then, within a week, he is likely to develop hemiparesis which is limited for the most part to the face and upper extremities. It is not the complete hemiparesis that occurs with lesions of the internal capsule. Very rapidly there develops a high leukocyte count, signs of meningitis, and a high spinal fluid cell count of leukocytes but no organisms in the spinal fluid on direct smear or on culture. The chief difficulty, of course, is to differentiate between such a diffuse subdural abscess and an acute encephalitis. It is perfectly justifiable and usually

necessary to make a diagnostic trephine in order to establish the diagnosis positively.

THROMBOSIS OF THE SUPERIOR LONGITUDINAL SINUS

Much that has been written regarding this condition as a clinical entity must be discarded. Formerly, it was supposed that paralyses of the extremities, would be produced by occlusion of this sinus posterior to the Sylvian veins. Some neurosurgeons now have no hesitancy in ligating the superior longitudinal sinus and in any location. The clinical picture of sepsis, delirium, upper extremity paralysis, and increased intracranial presure, ending fatally, and demonstrating at necropsy a thrombosis of the superior longitudinal sinus is probably due to a diffuse cortical thrombo-phlebitis. The end result of such vascular infection might well be a thrombus in the superior longitudinal sinus since this sinus may become thrombosed as the result of extension of infection from its tributaries. Primary thrombosis of this sinus is very rare in adults, perhaps because of its anatomical structure. It has an exceptionally heavy covering of dura and is extremely small where it is in close proximity to the frontal sinuses. Although I have seen and operated upon many cases of osteomyelitis of the skull in which there was a large midline epidural abscess overlying the superior longitudinal sinus, I have not seen a single case in which there was clinical evidence of a septic thrombosis of this dural sinus.

CAVERNOUS SINUS THROMBOSIS

A detailed discussion of this condition is not called for on this occasion. The diagnostic criteria of the fully developed case are well known. The decrease in morbidity and mortality in recent years is common knowledge. I wish only to remark upon the difficulty of diagnosis in the early stages when the clinical picture may indicate only a severe orbital cellulitis. Selective neurogenic extra-ocular muscle palsies rather than complete orbital fixation, signs of slight meningeal irritation, and profound sepsis characterize cavernous sinus thrombosis in its early stages. Papilledema is not diagnostic. In the late stages, of course, involvement of the contralateral side is almost pathognomonic. It has been suggested to me by Dr. E. A. Kahn that the presence of retinal venous pulsations in the uninvolved eye would tend to speak against the diagnosis of cavernous sinus thrombosis.

LEPTOMENINGITIS

Like infections of the brain substance, leptomeningitis may occur very early in the course of an acute suppurative sinus disease as the result of septic thrombo-phlebitis of communicating veins, or it may be the late result of a slower spread of infection by contiguity of tissue. The signs of the fully developed case are classical. I wish only to remind you that all of the intracranial complications may produce some signs of meningeal irritation which indicate their presence.

There is one type of septic meningitis complicating acute sinusitis which is rare but worthy of note at this point. An individual who has had a compound skull fracture resulting in cerebrospinal rhinorrhea may have spontaneous cessation of the flow of fluid. In case a funnel of dura and arachnoid becomes pinched between the bone fragments of the inner table of the frontal or ethmoid sinuses the patient has an opportunity to develop a purulent leptomeningitis complicating a rather mild attack of acute sinusitis. After such a patient has recovered from his acute meningitis, a repair of the dural defect must be accomplished in order to prevent recurrence of the accident.

FRONTAL LOBE BRAIN ABSCESS

In most clinics, as in the one with which I am associated, brain abscess like diffuse subdural abscess and purulent encephalitis is a condition finally diagnosed and treated by the neurosurgeon. There are certain generalities in the diagnosis of this condition which all rhinologists should keep in mind, however. I wish to emphasize the well known fact that the frontal lobes are largely silent areas. The focal signs of frontal lobe space occupying lesions are not prominent although very commonly a slight contralateral weakness of the lower facial muscles is noted.

One should entertain the thought that a frontal lobe abscess might be present if the patient remains ill with clinical evidence of undrained pus after the acute sinus infection is obviously under control as the result of medical or surgical treatment.

Differential diagnosis from clinical examination alone among frontal lobe abscess, subdural abscess, and even epidural abscess may be impossible. Of course, there is much less clinical evidence of an infectious process in epidural abscess than in subdural or brain abscess.

Diagnostic trepanation of the skull, ventriculography, and finally exploratory tapping may be required before a positive diagnosis can be made.

In conclusion, I wish to comment briefly on the surgical treatment of acute suppurative frontal sinusitis when there is evidence of an associated intracranial complication. If there is clear-cut evidence of a frontal lobe abscess, this should be attacked first, preferably by repeated tapping and finally by excision at the optimum time. During convalescence, a complete external frontal and ethmoid sinus exenteration should be performed which must include removal of the posterior wall of the frontal sinus in order to drain any epidural collection of pus which may be present.

If there is evidence of an intracranial extension of infection but the exact nature of this is not known, the sinus operation should be done immediately. Again, the posterior wall of the frontal sinus must be removed to permit thorough study of the underlying dura. Finally, in my opinion, the only acutely infected frontal or ethmoid sinus that should be operated upon is the one with evidence of extension of infection to the calvarium or intracranial contents. When an operation is performed in such an acute situation, it must be radical and complete. A timid surgical approach at this time invites disaster by promoting further extension of infection to the diploic structure of the skull and by leaving untouched the intracranial infection which may well prove fatal if drainage is not accomplished.

XLI

DISCUSSION

HARRIS P. MOSHER, M.D.

MARBLEHEAD, MASS.

Dr. McMahon

Dr. McMahon states that irregularities of the nasofrontal duct may be a protection instead of an invitation to infection of the frontal sinus. This is a new idea to me. He is generous in quoting the work of Mosher and Judd; and Furstenberg is given due credit.

He emphasizes the size of the diploic veins and their intimate connection with the veins of the frontal sinus. In dealing with acute infection, he emphasizes mostly changes confined to the mucous membrane. In chronic infection, I have always stressed that the fundamental element was an infectious thrombophlebitis. I still feel that way.

He lays stress on the fact that mucous membrane changes can be reversible. This has to be; otherwise, there would be no spontaneous cures, as often happens.

Dr. Goodale

TREATMENT

Conservative shrinking of the tissues in the anterior part of the middle meatus often is sufficient in acute frontal sinus infection. A more efficient and quicker method is to open the floor of the sinus above the inner canthus of the eye and irrigate the sinus with a solution of penicillin. The floor of the sinus has its weakest spot above the inner canthus of the eye.

THE LYNCH OPERATION

Dr. Goodale does not think the Lynch operation is successful in a large, shallow sinus. I agree. I think it stands a poor chance of success in a large sinus with many partial partitions, especially partitions near or at the outer angle. Dr. Goodale advocates, in radical operations, the removal of all the mucous membrane. This is hard to do in the

Lynch operation. Bits of mucous membrane not removed lead to pyoceles, and require a second operation.

Dr. Goodale has had success in keeping the nasofrontal duct open by a strip of tantalum foil. This appeals to me and warrants further trial and evaluation. He pays his respects to gold tubes to keep the enlarged nasofrontal duct open. This is a very old procedure, but it is used even today. It does not measure up to my ideal of surgery, which is to cure the patient and not make him a life-long patient. If both frontal sinuses are chronically infected, Dr. Goodale would operate on both sinuses and take away the inter-frontal partition. This helps obliteration, if that is hoped for. Deformity after a radical frontal sinus operation is no longer feared as it can be corrected by a bone graft, a tantalum plate or a thin plastic plate. The last-named is easier to manage than tantalum.

He says nothing about necrosis of the bridge in the Killian operation. In a number of my cases this has happened, with recurrence of symptoms.

Dr. Maxwell

A statement of Dr. Maxwell's which deserves a headline is: "An intracranial infection may complicate an acute frontal sinusitis even before an actual empyema of the sinus has had time to develop." Another similar one reads: "The otologist must not forget that he is a physician first," and I would add he must think beyond his specialty for complications.

In acute osteomyelitis Dr. Maxwell advises waiting for X-ray evidence of bone destruction. This means waiting a week. If the patient is acutely ill, I prefer doing an exploratory operation earlier than this.

Epidural abscess is such a constant companion of osteomyelitis of the frontal bone, it should always be excluded by a sufficient uncovering of the dura.

Epidural hemorrhage is new to me. I am grateful to Dr. Schneider for allowing Dr. Maxwell to tell us of his two cases of this rare condition.

I learned quite a few years ago from Dr. Kubik, Head of the Neurological Department of the Massachusetts General Hospital, about diffuse subdural abscess. The infection is generally associated with infection of the sinuses, especially the frontal sinus. The greatest

accumulation of the subdural pus is in the temporal fossa. If a trephine opening there does not locate it, a second or third opening should be made further forward, and nearer the frontal sinus. The characteristic of this type of infection is that the patient is quickly gravely ill, and gets progressively worse to an early death.

I have often wondered why in fulminating osteomyelitis the superior longitudinal sinus so often escapes. I have watched for this condition, but have never found it. As the motor centers for the legs touch the sinus on both sides, paralysis of a leg should be one of the first signs. The arm centers follow below the leg centers. Dr. Maxwell puts paralysis of the arm as the commonest form of paralysis in thrombosis of the superior longitudinal sinus.

Dr. Maxwell's paper is especially valuable both for the clear tabulation of the signs and symptoms of the intracranial complications of sinus infection, and for the frequent observations drawn from personal experience. They show a full knowledge of the general medical and the surgical backgrounds necessary for the successful treatment of these life-and-death cases.

I like the summary of his paper. I don't like giving to the brain surgeon—or, rather, having him grab—brain abscesses arising from infection in the frontal sinus or from the ear. We should make ourselves capable of operating them by the most up-to-date methods. At least, we can tap and tap, and wait and wait. This method has never appealed to me, but it has successes to its credit.

Dr. Goodale finds the treatment of acute frontal sinusitis simple and satisfactory and, naturally, goes on to the treatment of recurring attacks, and also to the operative treatment of chronic infection. This gives me a chance to speak of two subjects on which I have decided opinions. The first is the question of opening the posterior wall of the sinus in acute infections when the anterior wall is necrotic; the second is when and how to obliterate the frontal sinus when it is judged necessary.

Now, a few words on the first topic.

In acute frontal sinusitis where there is necrosis or perforation of the anterior wall of the sinus, microscopic examination of speciments removed at operation show that the posterior wall also shows infection. Therefore, I feel that it should be a rule to remove enough of the posterior wall to exclude extradural abscess. These abscesses are

often what might be called silent abscesses, but they are a frequent cause of osteomyelitis and intracranial complications.

On the second topic I have this to say:

Obliteration of the frontal sinus should be considered after a second operation, a so-called "revision", has failed. A revision is usually a hunt for mistakes in the first operation. Re-operations, euphoneously called "revisions", should not continue indefinitely. Dr. Schall quotes the case of a boy who had 17 operations. Naturally, these were not done by him.

The term "obliterating operation" is often a misnomer, especially when applied to the Killian type of operation because this has the mean habit of not obliterating. The frontal sinus operation for chronic suppuration is still a gamble as to a successful result.

The method of obliterating the frontal sinus which appeals to me is, as follows:

The basic idea goes back to my experience with osteomyelitis of the frontal sinus and the frontal bone. The technique of the operation is, as follows: Remove the whole of the anterior and posterior walls of the frontal sinus. Leave the floor intact. Leave the nasofrontal duct alone, or pack it with a gelatine sponge soaked in thrombin. Open the anterior ethmoidal cells, but not the posterior cells unless the X-ray shows marked disease there.

Leave the inner half of the skin incision open so that all parts of the sinus can be reached and packed, especially firm packing over the nasofrontal duct and at the outer angle of the former sinus as pockets are liable to form at these two places. Let the granulations from the dura and the bone edges of the removed sinus walls work to the surface and gradually obliterate the cavity of the former sinus. When repeated packings by coaxing the granulations and the accompanying fibrous tissue have done their best to obliterate the sinus cavity, close the skin incision. Then, at an appropriate time, correct the resulting deformity by an implant of a tantalum plate, or by a plastic plate.

Should both sinuses be operated upon at the same time, the inter sinus partition is no longer an obstacle to obliteration, as it is when only one sinus is operated upon, and the skin and underlying muscular tissue will lie flat against the dura and no packing is necessary—only

firm pressure from the surgical dressing. There is more deformity, of course, but this is corrected in the usual way.

The above method appeals to me as straightforward surgery.

In the cases of osteomyelitis of the frontal bone and the frontal sinus which I have followed, the ethmoid cells were left for a further operation, but as far as I know to date, an ethmoid operation in these cases did not prove to be necessary. This seems to show that taking care of the infection in the frontal sinus took care of the infection in the ethmoid cells. It suggests that we may have been putting the cart before the horse, that the frontal sinus infected the ethmoid cells and not the other way round, as most of us have long supposed.

CHEMOTHERAPY

Each of the foregoing papers naturally takes up chemotherapy and gives it due credit. Dr. Maxwell gives it a little too much. He makes a headline statement when he says: "The lack of a definite favorable response within 72 hours indicates inadequate therapy, and should prompt complete re-evaluation, including bacterial culture". The drawback to chemotherapy is that it often blurs the diagnosis of beginning complications, and often leads to watchful waiting in resorting to surgery. At the Infirmary in Boston recently there have been at least three cases where fulminating osteomyelitis steadily advanced in spite of heroic chemotherapy.

Before chemotherapy the antigens of the blood and the phagocites were alone on the job. Since the advent of chemotherapy we have greater reason to use and appreciate the phrase "The Good Earth", as moulds often hark back to the earth.

Dr. Goodale states that one of the first things to find out in infection of the frontal sinus is the type of the bacteria causing it. Dr. Maxwell gives the impression that a routine office culture is a bit of a chore. It shouldn't be. An office patient deserves as scientific treatment in this respect as a hospital patient.

Besides penicillin, we now have aureomycin and streptomycin. Two of these and, at times, the three, are used together. With Influenza B. there is a potent antitoxin.

Davison has written a valuable paper on the subject of sensitivity. If a case does not respond promptly to the drug employed,

the determination of the sensitivity of the causative bacteria to this drug should be settled at once. This, I admit, is not an office procedure. It is the most vital point in the use of chemotherapy. It does away with the old method of trial and error. It is one of the most important subjects dealt with in the papers just read. I almost added, "Would it not be useful if there were some such test for the good and bad in human nature?"

XLII

CHOICE OF TREATMENT IN CANCER OF THE LARYNX

YEAR 1949

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NEW YORK, N. Y.

CHOICE OF TREATMENT IN CANCER OF THE LARYNX

Malignancy of the larynx presents a dual problem to the laryngologist: (1) diagnosis, and (2) treatment.

Diagnostic procedures, fortunately, are well defined and offer no serious problem. However, once the diagnosis is established, the choice of treatment is not clear-cut and places a heavy burden of responsibility on the laryngologist. Shall it be surgery or radiotherapy, or possibly a combination of both? Which avenue will provide the individual patient with greater safety and more assured possibility of "cure"?

Before making a decision, the laryngologist must weigh many factors: size and location of the growth, site of its origin, the mobility of the cords, degree of involvement and whether or not the glands of the neck are involved. In addition to these physical and physiological factors, the laryngologist must also assay the emotional and mental attitudes of the patient and his future means of earning a living. Today, economic and social considerations must be regarded fully as important as the physiological factors. The aim must be not only the extirpation of the malignant growth but also the maintenance of the patient as an entity in society, with his economic and social responsibilities kept as normal as possible.

There was a time, some ten or fifteen years ago, when surgery was recognized as the only treatment in eradicating laryngeal cancers. But in those fifteen years, the science of radiotherapy has progressed to a point where today it must be realized that surgery is no longer the sole resort of the laryngologist. Trained radiotherapists are

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From the Manhattan Eye, Ear and Throat Hospital, New York, N. Y.

available. Accurate measurement of roentgen units are possible and clinical methods of application of radiotherapy have been developed.

Today, laryngologists realize that radiotherapy, often in conjunction with surgery or alone, has materially added to the possible "cures" of cancer of the larynx.

Present day methods of radiotherapy stem largely from Coutard's protracted fractional dose technique of deep x-ray, and his presentation in 1922 of a series of cases of laryngeal cancer cured with that type of therapy.

Basically, the principle is destruction of the growth with the least possible damage to normal tissue. This is accomplished by administering the treatment in fractional doses over a period of time which ultimately delivers the desired effective dosage. The end result is eradication of the growth without destruction of normal tissues. To employ this technique effectively, it is essential to realize the exact anatomical location of the lesion, its extent, and the mode of spread in each instance. With these factors known, it is possible for a trained radiotherapist to determine the total dose to be administered and select the method of application and the daily dose. Careful periodic examinations of the area during and after completion of therapy will disclose any evidence of possible recurrence.

Such a procedure obviously calls for the cooperative efforts of the laryngologist, the radiologist and the pathologist. Each individual case must be regarded as an individual problem.

There are, however, certain general factors which can guide us in choosing the selection of therapy. The site and origin of the growth, its extent and the general physical conditions of the patient, all play an important role. The microscopic structure of the growth is a factor but not a decisive one. For instance—though lack of differentiation in an epithelioma is usually regarded as a sign of radio-sensitivity, an undifferentiated but extensive and invasive epithelioma, is less radio-sensitive than a well differentiated one in the same location, but which is smaller and papillary in character.

We treat our patients daily for a period of approximately six weeks. Two lateral fields are employed and both are treated simultaneously. The preliminary x-ray beam is centered over the site of

the lesion. It is of the utmost importance to accurately localize each field. The entire lesion, plus a margin of normal tissue around it, must be included within the field of irradiation. The technique generally employed is as follows:

200-KV, 18 to 25 Ma.
½ to 1-mm. cu. filtration
50 cm. TSD, daily dose beginning with 75r., increase to 100r.

The size of the fields vary, decreasing slightly. For example, if we start with 6x8 cm., it will decrease to 6x6 and ultimately to 5x5 cm. A careful larynx examination, using laryngeal mirror, is made daily. At the height of the reaction, treatment is either discontinued for a few days or the daily dose is decreased. In our experience, we have found that 6,000 to 8,000 roentgen units are usually required to produce clinical cure of a squamous cell epithelioma.

The diseased tissues must be entirely included in the area which is adequately irradiated. If that is not done, the process outside of this zone, eg., cancer may progress in spite of satisfactory regression of the adequately irradiated portion. Accurate placement of the beams of radiation is of paramount importance. For this reason, radiotherapy must be done by, or under the direct supervision of a physician who has clinical judgment, radiotherapeutic experience and is familiar not only with the technical part of radiotherapy but also with the clinical behavior and anatomical location and extent of the diseased process. The radiotherapist himself, must be able to examine these anatomic structures in order to detect radiation changes and adjust individual radiation doses accordingly.

The radiotherapist, treating laryngeal cancer, must specialize in this field just as the surgeon must be trained and qualified in this branch of surgery. He should be able to examine the larynx with a head and laryngeal mirror.

First, to plan the course of therapy, choose the size of the portals and dosage. In early carcinoma of the cord, very small fields are irradiated to prevent unnecessary edema and late changes. On the other hand, if one is dealing with a carcinoma arising in the epiglottis, larger portals are chosen in order to include the channels of lymphatic spread.

Secondly, it is important to examine the larynx at frequent intervals during the course of treatment, watching for edema and expected pseudo-diphtheritic membrane. Very often the daily dose has to be cut down or therapy has to be interrupted for a while and the size of the portal is gradually cut down as the lesion regresses.

Follow-up on the cases is also important to determine whether observed edema is due to irradiation or a sign of residual disease. Surgery occasionally follows radiotherapy. If proper irradiation is given, there is little delay in healing when cases do become operable.

When the growth is observed early and is small, involving one cord which is still freely movable, it may be removed by simply biting it out with a cup forceps through a laryngoscope and fulgurating the base with electric current as demonstrated by Le Jeune and New. This type growth is also responsive to rediotherapy. Irrespective of what type of treatment is employed, these cases must be carefully followed so as to make certain that there is no future recurrence.

When the growth is larger, involving one cord but not extending into or across the anterior commissure, or not extending posteriorly further than the vocal process with a freely movable cord and no subglottic extension, laryngo-fissure is the operation of choice which gives an 80% cure. But here again, radiotherapy seems to be as effective as surgery with as good a five year cure rate.

Until a few years ago, I must admit to some skepticism about the effectiveness of radiotherapy for these type cases. Approximately ten years ago I examined three patients on whom I advised laryngo-fissure. All three refused and had radiotherapy instead. Today, they are alive and well with practically normal voices. Subsequently, from my clinic at the Manhattan Eye, Ear and Throat Hospital, I referred a number of these early cases for radiotherapy. At the present time 33 such patients have been subjected to radiotherapy with the following results:

2 patients died of local persistence of the disease—(refused surgery)

2 patients died of metastasis—(1 lung, 1 skull)

2 patients died of other causes—(circulatory)

2 patients had persistence of the growth after radiotherapy and subsequently underwent laryngectomies. They are alive and well after 3 years.

Of these 33 cases—27 patients are living and well. Of these one is over 11 years, one over 8 years, one over 10 years, one over 7 years, five over 4 years, five over 3 years, five over 2 years, three over 1 year, 11 under 1 year.

For the past five years at the Manhattan Eye, Ear and Throat Hospital, we have been trying to divide these early cordal cases equally between radiotherapy and surgery for a comparative study of end results, which I hope to report at a later date.

When the growth is in the posterior portion of the larynx or where there is fixation of the cord or subglottic extension or involvement of the aryepiglottic fold or pyriform sinus, laryngectomy is the treatment of choice, followed by x-ray therapy. In these cases, laryngectomy gives a much higher clinical cure rate than roentgen therapy.

In cancer of the epiglottis where the growth involves the tip, provided it has not invaded the posterior half, the epiglottis may be removed without disturbing the rest of the larynx, either anteriorly through the neck or by suspension laryngoscopy. If the growth is a papillary, exuberating, bulky one, which only slightly invades the deeper tissues, it usually responds to x-ray therapy. These papillary tumors and their cervical metastases are radio sensitive and the metastatic glands should be included in the field of irradiation for the primary lesion.

On the other hand, where the growth tends to grow inward, involving the posterior half of the epiglottis, the larynx should be removed and x-ray therapy should follow.

Cancer of the ventricular band is not very common. The few that have been seen in the past few years have done well with x-ray therapy. In these cases, we believe that they should be treated by x-ray and carefully watched. If there are signs of recurrence, laryngectomy is advisable.

When the growth originates in the pyriform sinus, laryngectomy should be done at once, followed by x-ray therapy. Block dissection should be used if the cervical glands are involved. X-ray

therapy alone, has been practically useless in this type lesion. Where the growth involves the aryepiglottic fold laryngectomy is the treatment of choice, although we have a few patients who refused surgery and who were apparently cured by x-ray therapy. The closer this type of lesion is to the epiglottis without extension toward the arytenoid, the more radio sensitive it seems to be.

Where there is extensive extrinsic involvement, such as in the cases which we called inoperable in former years, I believe the treatment of choice should be a full course of x-ray followed by laryngectomy. Occasionally we are surprised to note how well these cases respond to x-ray and how easy it is to remove the larynx afterward, if necessary. In the past, when large fields and too intensive dosage were used, it was almost impossible to differentiate the tissues of the neck if a subsequent laryngectomy had to be performed. Today, with the divided doses and small fields, it is a simple matter to remove the larynx.

In clinical metastatic cervical lymph nodes, secondary to primary lesions of the vocal cord or subglottic region, block dissection of the cervical chain is indicated. When these metastatic nodes are secondary to primary lesions of the epiglottis or false cords, x-ray therapy is the preferred treatment. Our results with metastatic nodes, secondary to carcinoma of the pyriform sinus or arytenoid, have been poor with either surgery or x-ray therapy.

Every case must be carefully followed afterwards. If x-ray therapy has been given and recurrence is observed, surgery can be done at that time.

The above remarks are not intended as a complete classification of laryngeal cancer and indications for therapy. They do, however, represent some of our experiences at the Manhattan Eye, Ear and Throat Hospital where we have been particularly fortunate in having the services of Doctor Maurice Lenz and Doctor Clara O'Krainetz who are both outstanding authorities in laryngeal radiotherapy.

Our experience, however, does emphasize the obvious advantages of a complete classification of laryngeal tumors, with indications for therapy for the guidance of all laryngologists. I do feel that our Society should appoint a committee to study this need with a view to formulating such a classification of laryngeal cancer as a guide to therapy.

Despite the fact that the individual case requires individual analysis and decision, such a classification would immeasurably aid the laryngologist in reaching a decision which would offer his patient the best chance for recovery and rehabilitation.

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XLIII

REMOVAL OF CARCINOMA OF THE LARYNX WITH IMMEDIATE SKIN GRAFT FOR REPAIR

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ROCHESTER, MINN.

In the treatment of malignant tumors of the larynx, the primary objective must always be to eradicate the neoplasm. Preservation of laryngeal function, maintenance of a normal respiratory tract and shortening of the period of healing, and incidentally of hospitalization, are matters of secondary consideration. Frequently, the most extensive surgical procedures feasible are required in order to completely remove cancer in this situation, but laryngectomy should never be performed when a more conservative type of operation will suffice. Moreover, when sacrifice of the larynx is necessary, it seems only rational to avoid any possibility of too limited excision and to carry out as wide a removal as is consistent with the pathologic condition and with satisfactory repair, rather than to temporize with such procedures as subperichondrial resection, as has been advocated in some quarters.

Immediate skin grafting to restore the lining of the larynx following removal of carcinoma of this structure was carried out in a small group of carefully selected cases. The purpose of the procedure was to effect removal of the malignant process by means of a more conservative type of operation than would have been required otherwise, and to facilitate healing. The lesion in each of the three patients treated in this manner was of such extent that laryngectomy would have been the only surgical alternative. In each instance the skin graft took perfectly, and healing was hastened considerably. The patients have now been free from evidence of malignancy for periods of one year, two years and four months, and two years, respectively, the airway is unobstructed and intact, and the voice ranges from very hoarse to almost normal.

Read at the meeting of the American Laryngological Association, San Francisco, California, May 23 to 24, 1950.

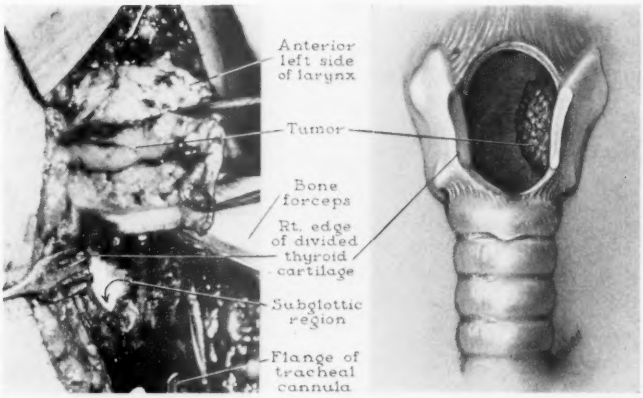

Fig: 1.—Left hemilaryngectomy (case 2). The two laryngeal forceps are grasping the anterior border of the left side of the larynx, which is being removed.

The field of usefulness of laryngofissure has expanded considerably during the past few decades, and the attitude of some eminent laryngologists that the operation should be limited to cases of early laryngeal cancer has changed completely. The operation has been made increasingly radical by supplementing sharp excision with electrocoagulation and by sacrificing more and more of the alae of the thyroid cartilage. Not infrequently the operation amounts to hemilaryngectomy, except that the continuity of the cricoid cartilage is not disturbed.

The extent of malignant tissue removable through a laryngofissure varies greatly, depending on the situation of the involvement, its nature, activity, extent, and previous therapy. It varies also with the training and experience of the physician. Generally, wider experience has demonstrated the feasibility of removing through a laryngofissure many malignant tumors of moderate activity or low grade, for which laryngectomy was previously performed. On the other hand, increasing experience has in some instances shown the necessity of wider removal of more active growths, particularly when there has been an associated inflammatory process, or the tumor was recurring.

The serious responsibility of deciding between conservative and radical therapy for malignant tumors of the larynx is borne entirely too lightly by some laryngologists. The physician should attempt to place himself in the position of the patient so afflicted and consider the drastic change in his mode of life that total laryngectomy will entail. The criteria applied by different laryngologists in determining when laryngofissure will no longer suffice for adequate removal of a malignant neoplasm and laryngectomy is advisable, vary greatly. Some surgeons perform thyrotomy only for freely movable lesions limited to the midportion of one vocal cord and recommend removal of the larynx when the carcinoma extends across the anterior commissure and involves the false cords or ventricle or when there is extension onto an arytenoid. Others remove through a thyrotomy a high percentage of lesions of this type even when there is limited mobility, providing the tumor is not highly malignant. In spite of this difference, the percentage of five-year survivals following the operation reported by experienced laryngologists shows remarkably little variation.

It frequently is impossible to determine definitely by clinical examination the limits of a malignant tumor of the larynx, and because of this it has for a number of years been the practice of my colleagues and myself to defer a decision concerning the surgical procedure necessary for its removal until direct inspection is made through a laryngofissure. Exploration of this type is advisable in all cases in which the growth is not well defined. Often it is possible to remove through this approach lesions that on indirect examination appeared to require laryngectomy. If direct inspection through the thyrotomy shows that the tumor is too extensive for removal by this route, laryngeal forceps are applied to the margins of the divided thyroid cartilage and laryngectomy is proceeded with.

In an effort to extend the limits of local resection for malignant laryngeal tumors without complete removal of the larynx, a few basic principles of plastic surgery have been applied. Among these is the precept that appreciable loss of surface covering or of the lining of a viscus must be restored in order to secure prompt and satisfactory healing and to obviate contracture; also, that a skin graft should not be used to cover a defect unless there is reasonable assurance that the pathologic process responsible for the loss has been completely eradicated. In addition, lessons learned in treating cic-

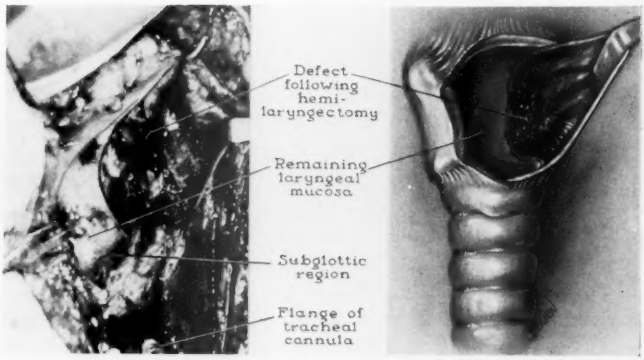

Fig. 2.—Larynx viewed from same angle (case 2) following hemi-laryngectomy.

atricial stenosis of the larynx by excision and skin graft have proved helpful in these cases.

The use of free skin grafts for restoring the lining of the larynx in cases of cicatricial laryngeal stenosis was introduced in 1930. Since then the procedure has been rather widely employed for this purpose, and numerous reports in the literature record almost uniformly satisfactory results. The operation consists essentially in re-creating a laryngeal lumen of adequate diameter by excising the stenosing scar together with any cartilaginous thickening, which may be a factor, inserting a moderately thin skin graft, and after the graft takes, keeping a dilator in place continuously until the graft loses its tendency to shrink. The skin graft should be cut sufficiently thin so that hair follicles will not be transplanted with it, and is preferably secured from a non-hair-bearing surface. It is held in the laryngeal wound by wrapping it about a stent of sponge rubber, which in turn is securely anchored in position by transfixing it and the laryngeal wound and soft tissues of the anterior portion of the neck with stainless steel wires. On the ninth to twelfth postoperative day the patient is anesthetized intravenously with pentothal sodium following thorough cocainization of the pharynx and upper part of the larynx, the larynx is exposed by suspension laryngoscopy, and the sponge rubber stent is removed through the mouth. At the same time an elastic or plastic dilator is placed in the larynx and left for

several weeks or months, depending on the tendency of the graft to shrink.

This procedure has at times been carried out in cases of cicatricial stenosis resulting from removal of cancer involving both sides of the larynx after complete healing of the wound has occurred. However, as far as I am aware, excision of laryngeal carcinoma has not previously been followed by immediate skin grafting of the wound. When a graft is applied at the same operation, the surgeon must, as has already been noted, be reasonably certain that the tumor has been completely removed. Such assurance can be gained only by careful consideration of the history, study of the lesion by indirect, and if necessary, direct laryngoscopy, critical inspection through an exploratory thyrotomy, and detailed microscopic examination by a competent pathologist. Careful check of fresh frozen sections of the peripheral portions of the tumor at the time of exploration assists greatly in determining accurately the limits of the growth. Judicious selection of cases is essential when immediate skin grafting is to be carried out following removal of epithelioma of the cutaneous surface. It is even more important in patients with laryngeal involvement because of the more serious possibilities, and the greater difficulty of accurate postoperative observation.

Healing usually takes place within a few weeks after removal of even a rather large epithelioma of the larynx through a thyrotomy. This occurs in spite of thorough electrocoagulation of the wound, although the healing process may be delayed for some weeks or months by the development of a granuloma or a sequestrum of cartilage. The same is true after hemilaryngectomy, except that more time is likely to be required. When an excessive amount of the ala of the thyroid cartilage on one side is taken away and removal of the soft tissue extends well beyond the midline, collapse of the laryngeal wall and cicatricial stenosis may result. In addition, there often is persistent edema of the aryepiglottic fold or ventricular band, especially if perichondritis is present. This edema often lasts a year or more and may necessitate wearing the tracheal cannula throughout this period.

When skin grafting was done immediately after removal of the carcinoma, cicatricial stenosis was obviated, and in the two cases in which most of one ala of the thyroid cartilage was removed, there

Fig. 3.—Skin graft wrapped about stent ready for insertion.

was no tendency for the laryngeal wall to collapse. In one of these two cases much less edema of the introitus of the larynx developed than usually occurs after such wide sacrifice of tissue, but in the other case a good deal of edema developed. The former patient, in whom a hemilaryngectomy was carried out, was decannulated on the twentieth postoperative day. He was breathing freely, and had a good, although hoarse voice, at the time of dismissal on the twenty-fourth day. The other two patients on whom early skin grafting was performed went home wearing a tracheotomy tube. In one of these the cannula was removed on his return to the Mayo Clinic about two and one-half months following the operation, the tracheal fistula closing spontaneously. In the third patient the anterior third of the left vocal cord, the anterior half of the right cord, the entire ventricle on both sides, and the anterior portion of both ventricular bands were removed, also approximately the anterior third of both thyroid alae. Because of this extensive sacrifice of tissue, severe laryngeal stenosis was anticipated, and an iodoform gauze pack was inserted to keep the incision open, actual insertion of the skin graft

being delayed until the twenty-first postoperative day. A plastic dilator was used in this case subsequently, and was worn for two and one-half months. Closure of the laryngeal fistula was not performed until nine months following the original operation because multiple small benign papillary growths developed in the posterior part of the larynx, and necessitated electrocoagulation through the laryngeal stoma on two occasions. When this patient was last observed two years after the initial operation, there was no evidence of recurrence of the neoplasm, the airway was free, and the patient had a good voice.

In using skin grafts to reline the larynx the possibility of transplanting hair follicles along with the graft must be borne in mind. However, this hazard is readily obviated by cutting the graft thin enough to avoid transplantation of hair follicles, and by utilizing a non-hair-bearing area as a donor site. Another disadvantage that occurs infrequently, but which at times causes considerable difficulty and annoyance, is the accumulation of sebaceous secretion and tenacious mucus on the surface of the skin graft, with the subsequent formation of foul crusts. The ozena resulting from this may be bitterly complained of by the patient. The liberal internal use of iodides, combined with frequent spraying of the larynx with saline solution and with oily preparations, generally is effective in combating the condition. Infrequently, removal of the crusts with laryngeal forceps is necessary (Figs. 1, 2, 3, 4 and 5).

REPORT OF CASES

CASE 1.—A man, aged 57 years, a storekeeper, came in on January 16, 1948, for consideration of progressive hoarseness of three years' duration. This had been much more pronounced for the past two months, and during the latter period he had experienced severe choking spells lasting from ten to fifteen minutes. His general health had been good, except for a recent attack of pneumonia. Examination revealed an obviously malignant lesion involving and fixing the left side of the larynx. There was no respiratory embarrassment, and no enlargement of the regional lymph nodes could be made out. On January 22, 1948, suspension laryngoscopy was carried out with the patient under intravenous pentothal sodium anesthesia for more detailed examination and biopsy. This showed the lesion involving the entire length of the left vocal cord, and the ventricular band, and producing considerable fixation. Tissue

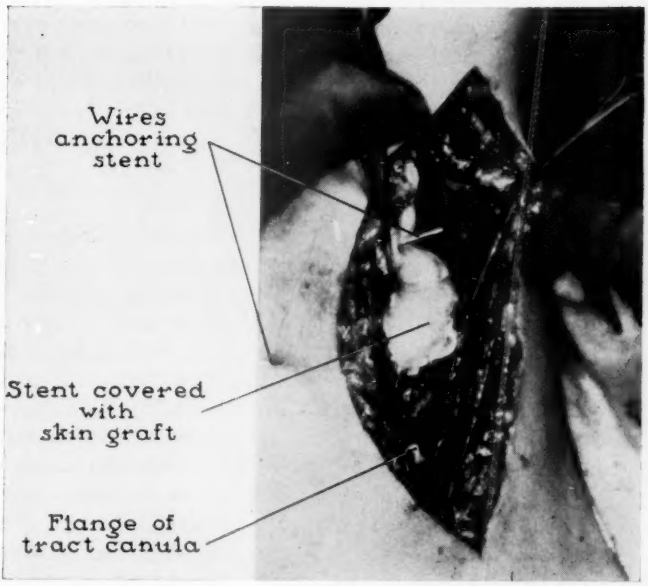

Fig. 4.—Stent supporting skin graft in laryngeal wound.

removed for microscopic study showed squamous cell epithelioma, grade 2 (Broders' method). Exploration of the tumor through a thyrotomy was advised, although it was anticipated that a laryngectomy would be necessary.

Two days later, using cervcial block anesthesia, the epithelioma of the larynx was explored through a thyrotomy. The growth involved the entire left vocal cord, the ventricle, and most of the ventricular band on this side, and was extending into the underlying muscle. Wide excision was carried out, removing most of the left ala of the thyroid cartilage along with the lesion, the procedure amounting to a hemilaryngectomy, except for the intact cricoid. The trachea was then opened, and after changing gloves, a moderately thin dermatome graft was removed from a hair-free area on the anterior abdominal wall. This graft was used to line the laryngeal wound, being applied about a sponge rubber mold, which was anchored in place by transfixing it together with the soft tissues of

the neck and the laryngeal wall of the opposite side with stainless steel wires. The ends of the wires were twisted together over the gauze dressing on the front of the neck, after closure of the neck wound. The procedure was much the same as that carried out in cases of severe cicatricial stenosis of the larynx in which excision of the stenosing scarring and relining of the airway with a skin graft are required.

The patient's postoperative course was not remarkable. There was no undue local reaction. On the third day after the operation the temperature rose to 101.8° F., then gradually subsided. On the tenth postoperative day the portion of the neck wound above the tracheotomy was healed, and using the suspension laryngoscope the sponge rubber stent supporting the skin graft was removed through the mouth. The skin graft lining the left side of the larynx had taken completely. The tracheal cannula was removed ten days later, and the patient returned home on the twenty-fourth postoperative day. At that time the larynx was in excellent condition, considering the extent of the operative procedure. There was much less edema than one would expect; the breathing space was ample, and the patient was able to talk aloud. Subsequent observation of this patient's larynx has been carried out at intervals of a few months, the last examination having been made on May 10, 1950. This was a little more than two years and four months following operation. At no time has there been evidence of recurrence of the malignant process, and the skin graft has given no trouble (Fig. 6).

CASE 2.—A man, 56 years of age, was referred by his home physician on May 30, 1949, because of progressively increasing hoarseness of eight months' duration. His general health had been good, and he had received no treatment for his throat condition. Indirect laryngeal examination revealed what appeared to be an epithelioma involving the entire length of the left vocal cord, and producing moderate limitation of motion. There was no palpable enlargement of the regional lymph nodes. General physical examination otherwise revealed no significant changes. Using indirect laryngoscopy and local applications of 10 per cent cocaine solution for anesthesia, tissue was removed from the lesion of the larynx in the office. Microscopic examination showed the growth to be a grade 2 squamous cell epithelioma. Exploration through a laryngo-

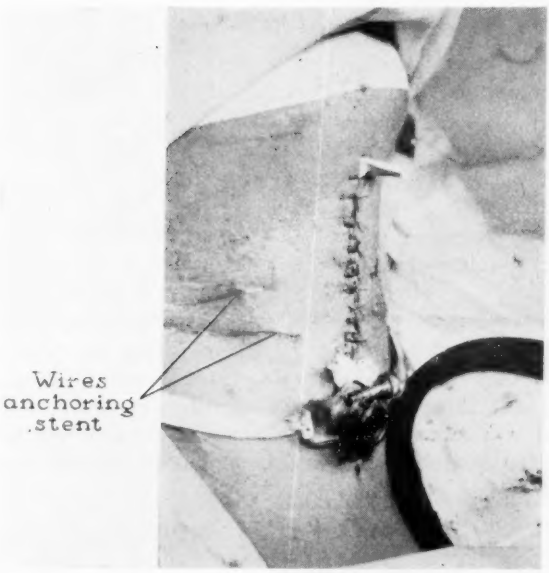

Fig. 5.—Neck wound following closure.

fissure was advised, although it was anticipated that a laryngectomy would be necessary.

The exploratory procedure was carried out on June 2, 1949, using regional (cervical block) anesthesia. Direct inspection of the growth thus afforded showed the malignant process involving the entire length of the left vocal cord and the floor of the ventricle, and extending into the ventricular band anteriorly. Grossly, the neoplasm looked more active than grade 2 squamous cell epithelioma, as it had been reported. There was only limited mobility, and the advisability of performing laryngectomy was seriously considered. However, it appeared that adequate removal could be effected by means of a hemilaryngectomy, and this was carried out after the trachea had been opened. Almost the entire left ala of the thyroid cartilage was removed, along with the growth. Immediately following this the wound was lined with a moderately thin dermatome graft removed from the right lateral abdominal wall. The growth was

supported by a sponge rubber stent, which in turn was anchored in place by means of stainless steel wires.

The patient's convalescence was uneventful. On the eleventh postoperative day the stent supporting the graft was removed through the mouth, using suspension laryngoscopy. At that time there was so much edema of the left arytenoid and aryepiglottic fold that only a portion of the skin graft could be visualized and the extent of the "take" could not be determined. The patient was sent home with the tracheal cannula in place on the eighteenth day following the hemilaryngectomy. He returned for observation a little less than two months later. He looked well generally. Indirect laryngoscopy showed some persistent edema of the left side of the introitus of the larynx, but no evidence of recurring epithelioma could be made out. Suspension laryngoscopy on August 16, 1949, showed no neoplasm present, and the skin graft was in excellent condition. The right arytenoid was quite freely movable. The airway was wide open. The tracheal cannula was removed, the tracheal fistula closing spontaneously within two days (Fig. 7).

CASE 3.—A man, 42 years of age, was referred for consideration on April 19, 1948, by his local laryngologist, who had discovered a growth in the patient's larynx. Indirect laryngoscopy revealed an ulcerated lesion involving approximately the anterior third of both vocal cords and the anterior commissure without apparent fixation. A biopsy performed in the office using local anesthesia and indirect laryngoscopy revealed grade 2 squamous cell epithelioma.

Exploration through a laryngofissure was carried out on April 29, 1948, using regional anesthesia. This was done without opening into the growth. The tumor was found to be much more extensive than it had appeared on indirect examination. It involved the anterior third of the left vocal cord and the anterior half of the right cord, and extended so far posteriorly in both ventricles that removal necessitated sacrifice of tissue almost back to the vocal process on both sides. Because of the depth of the involvement, the anterior third of the ala of the thyroid cartilage on both sides was removed. The tumor presented a papillary structure and was multicentric, there being numerous small papillary lesions extending well posteriorly on both vocal cords, in the ventricles and on the right ventricular band beyond the main mass. These were thoroughly electrocoagulated. After the trachea had been opened, the laryngeal wound

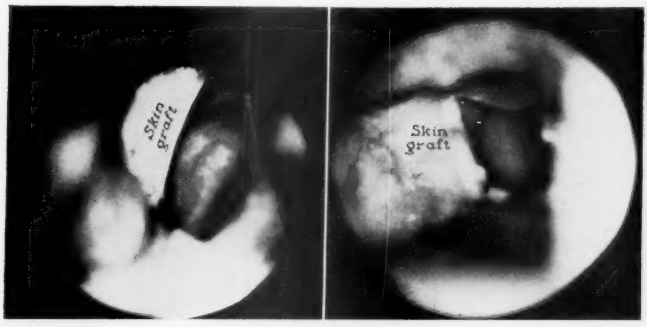

Fig. 6.—View of larynx about two years following hemilaryngectomy and immediate skin graft (case 1).

Fig. 7.—View of larynx two and one-half months following hemilaryngectomy and immediate skin graft (case 2).

was packed open with greased iodoform gauze, for it was appreciated that marked cicatricial stenosis of the larynx would almost certainly develop as healing occurred. It was anticipated that relining the larynx with a skin graft after some months would be necessary, and that retention of the tracheal cannula in the meantime would be required.

Postoperatively so little local reaction developed that it was decided to line the laryngeal wound with a skin graft, rather than wait for stenosis to take place, and then correct the condition secondarily. Accordingly, on the twenty-first postoperative day, the iodoform gauze pack was removed, the granulations were curetted away, the wound was dusted with penicillin-sulfanilamide powder, and a moderately thin skin graft was applied about a sponge rubber stent as in the previous cases.

On removal of the stent nine days later, the graft had taken perfectly. A plastic obturator was fitted, and the patient returned home on the thirty-fifth day following removal of the laryngeal carcinoma. He returned for observation a little more than two months later, at which time examination showed multiple small papillary growths on the posterior portion of the left vocal cord and the left arytenoid. Microscopically these showed no malignant change, but thorough electrocoagulation was carried out through the lar-

yngostomy. Repetition of this procedure was again necessary on the patient's return two and one-half months later. Further observation on February 3, 1949, a little more than nine months after the initial operative procedure, showed no evidence of recurrence of the neoplasm, and the airway was ample. Accordingly, plastic closure of the laryngeal fistula was carried out. Subsequent observation has shown no recurrence of the laryngeal pathologic change, the last observation being two years following the initial operation. The patient is in excellent general health, has a good voice, and is working right along.

CONCLUSIONS

Three cases have been reported in which rather extensive malignant tumors of the larynx were removed by hemilaryngectomy followed by immediate skin grafting for repair. The lesions were of such extent that laryngectomy would have been indicated had skin grafting not been carried out. The period of time following removal of the malignant process is too brief to warrant a statement concerning control of the disease; however, the progress of the patients to date well justifies further trial of the procedure.

MAYO CLINIC.

XLIV

REHABILITATION OF THE LARYNGECTOMIZED PATIENT

LeRoy A. Schall, M.D.
Boston, Mass.

(Motion Picture with Sound)

XLV

THE SURGICAL CORRECTION OF CICATRICIAL STENOSIS OF THE LARYNX

FLETCHER D. WOODWARD, M.D.

CHARLOTTESVILLE, VA.

The treatment of cicatricial stenosis of the larynx has always been a difficult problem, and still taxes the ingenuity of the laryngeal surgeon. However, as time has gone on, the principles of treatment have been evolved to such an extent that one accepts such a case today with less trepidation than formerly.

The number of such cases is less, although their severity is often greater. This reduction in number is largely due to the efficiency of medical prevention and treatment of diphtheria and syphilis, to improvements in radiation technic, to the dissemination of Chevalier Jackson's repeated warnings against high tracheotomy, and to the better diagnosis and treatment of infectious processes of the larynx with the antibiotics. The increase in severity is largely due to trauma, incident to high speed transportation and war wounds and, occasionally, to surgical procedures. The widespread use of intratracheal anesthesia, especially in the hands of unskilled anesthetists, may be a factor in the production of such cases in the future.

Repeated dilatation, both peroral and retrograde, with various types of dilators, the use of rubber tubes attached to the tracheotomy tube, and the Jackson core molds have cured many cases. But these methods all require much time and patience on behalf of surgeon, and time and discomfort for the patient; and they present quite an economic problem, especially for those patients who live some distance away. Jackson, Foster, Jesberg, and others have reported many successful cures by these methods.

The open approach with the insertion of an indwelling rubber tube was popularized by von Schmiegelow, and many severe cases have been successfully treated in this manner. Refinements in the open approach technic, especially when combined with skin grafting and the use of an acrylic resin mold, have been made by Arbuckle,

Le Jeune, Figi, Erich, and Looper, who also used the hyoid bone itself as a graft, in order to gain additional space in certain cases.

I have never been quite satisfied with any of these methods. There was always something to be desired, either in the method, my ability, or both. In repeated bouginage, the unsatisfactory features were: the time required, the patient's discomfort, and the economic problem. In the use of the rubber indwelling tube, the undesirable features were frequently: the secretion and odor from irritation, the formation of granulation tissue, and the subsequent alteration in the length and diameter of the tube. In skin grafting, there was the necessity of maintaining a large stoma, the removal of the stent, the difficulty of getting an impression for the acrylic resin mold, and finally the plastic operation required to close the stoma after an adequate airway had been obtained. (For those who prefer this method, I would like to mention that the impression compound called "Sonotone Mix," prepared by the Sonotone Company for making ear molds, has been most satisfactory and superior to dental compound).

For these reasons, I made an effort to combine certain features of each method for clinical trial. Desiring a nonirritating tube rather than a solid mold, I had the larger sized O'Dwyer intubation tubes duplicated in an acrylic resin, and, following the suggestion of Robb, barium powder was incorporated in the plastic to make them radiopaque. Holes were drilled in the lower end, middle, and upper end, for the insertion of tantalum or steel fixation wires and traction sutures if desired. These tubes have the proper shape, are nonirritating, will not change shape, can be trimmed at the operating table with the motor-driven saw, can be removed perorally, are radiopaque, and can be used, if desired, according to the core mold technic of Chevalier Jackson. The lumen is sufficiently large to permit breathing and speech; and if the tube should become dislodged and pass below the tracheal cannula, an adequate airway will be furnished through and around the tube.

Since the stenotic area in many cases is not extensive, sharp submucous dissection of the cicatricial area was substituted for skin grafting, and experience has shown that epithelization takes place rapidly enough to prevent contracture. This obviates an operation for the removal of the stent and, finally, one for plastic closure of the necessarily large stoma.

When there was a loss of the cartilaginous framework, a free graft from the central area of the hyoid bone was employed between the separated alae of the thyroid cartilage. This graft seemed well suited by its shape and nature, and was readily obtained and maintained in its new position by chromic gut suture.

This one-stage method has now been employed successfully in four cases. It requires a short period of hospitalization. The patient can return home for six months—as is required in some cases—and follow his vocation, can breathe fairly well naturally, can talk, and is comfortable. Finally, the tube can be removed perorally, and the small tracheotomy opening will either close spontaneously or can be closed by a very simple plastic procedure under local anesthesia, after an adequate lumen has been obtained.

The degree and extent of the stenosis can be determined quite accurately by mirror and direct laryngoscopy, by lateral neck roentgenograms occasionally with iodized oil, by tomography, and finally by inspection from below by retrograde tracheoscopy with the open tube, or by the nasopharyngoscope introduced through the tracheal opening.

If the stenosis is due to tuberculosis or cancer, one must be sure that the disease is arrested, before operating. In traumatic cases, a bilateral recurrent nerve paralysis is possible, and may require an arytenoidectomy subsequently, unless an adequate glottic airway can be obtained at the primary operation in addition to an adequate laryngeal airway. Finally, in cases due to a recent high tracheotomy, merely lowering the tracheal tube to a new position may be sufficient to restore an adequate lumen.

The operation is carried out under local and sodium pentothal anesthesia. The thyroid cartilage and, if necessary, the cricoid and upper trachea are opened in the mid line. A motor-driven saw is frequently required for this step. The cicatricial tissue is removed by sharp dissection, and the mucosa is preserved as much as possible. If there has been no loss of cartilage, a graft is unnecessary. A suitably sized mold is selected, trimmed to fit as well as possible, and its position maintained by relatively stiff tantalum or steel dental wire. This wire is passed through the holes in the mold, knotted once, and its free ends passed through the thyroid cartilages; they are then bent down close to the cartilage and remain in this subcutaneous

position. This prevents the mold from passing down the trachea, yet these ends will trail when the mold is removed perorally.

If the continuity of the cricoid ring has been destroyed, it should be restored as well as possible if there has been a loss or marked distortion of the cricoid or thyroid cartilage; then a graft is obtained from the center of the hyoid bone and sutured between the wings of the cartilage, with chromic gut suture. Small holes punched in the edges of the graft facilitate this step. These grafts have remained viable; and when the antibiotics are used, both pre- and postoperatively, we have had primary healing. Following the operation, the position of the tube can be verified at any time by indirect laryngoscopy and by roentgenograms. The mold should be allowed to remain in situ from 2 to 6 months, depending upon the degree and extent of the stricture. Since these patients are comfortable and can talk and breathe quite well, it is better to leave the mold in too long rather than take a chance on cicatricial contracture by removing it too soon.

There has been no loss of sphincteric muscle action, in these cases, but there is impaired voice in those in whom the graft has been used; however, none of these patients have complained of this. Their desire for an adequate airway and decannulization is paramount to a good voice, especially in those who have had a complete stenosis and were therefore aphonic.

Although one cannot draw many conclusions from a small group of four cases, the procedure outlined in this paper has given better results for me than any I had tried previously. Nothing new has been offered. I have merely combined certain features of the many operations advocated, by various surgeons, into a one-stage procedure and if further experience justifies my present enthusiasm, I shall continue to advocate it for all such cases, as the easiest and simplest way to correct a serious and difficult problem.

CASE REPORTS

CASE 1.—A laryngofissure for early cordal cancer, grade 1, was done on a woman by the Kemler technic; and although this method had been successfully employed in several men previously, I did not take into consideration the shorter anteroposterior diameter, nor her tendency to keloid formation. As a result, I was unable to decannulate her. Several months later, the larynx was reopened.

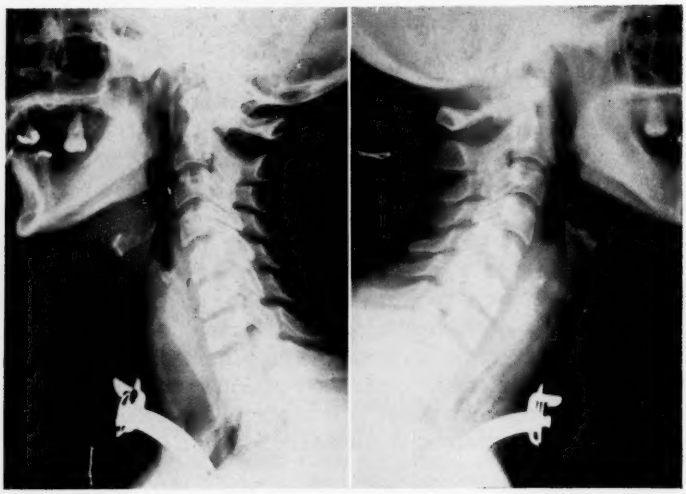

Fig. 1.—Preoperative lateral roentgenogram of neck, showing complete stenosis.

Fig. 2.—Postoperative lateral neck roentgenogram, showing plastic mold in situ and hyoid bone graft.

A free cartilage graft (Bone and Cartilage Bank) was inserted, between the thyroid cartilages, to give added space, and an acrylic tube inserted. An adequate airway was obtained, but the voice was poor.

CASE 2.—This patient had a complete stenosis, of many years' duration, from an inflammatory process of unknown etiology. At operation the cicatricial tissue was excised and an acrylic tube and a hyoid graft inserted, because of the very small laryngeal lumen. The tube was worn for six months; it was then removed perorally, and an adequate airway obtained. The voice is fair, and the old tracheal fistula was closed by a simple plastic procedure under local anesthesia.

CASE 3.—This patient had had a high emergency tracheotomy performed elsewhere; and since her doctor was unable to decannulate her, she was referred for operation. On her first admission, the tracheal tube was lowered to the suprasternal notch, and her airway was much improved in six weeks. However, it was inadequate; so the larynx, cricoid, and upper trachea were opened. It was noted

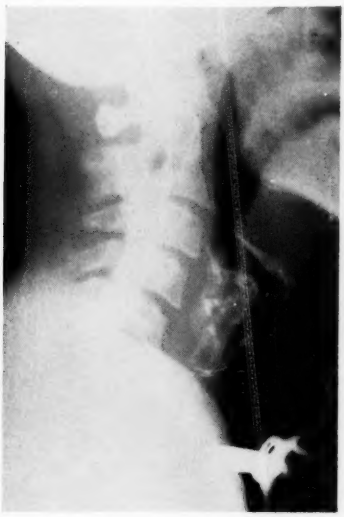

Fig. 3.—Postoperative lateral neck roentgenogram. Note radiopaque plastic mold, hyoid bone graft, and tantalum wire.

that a part of the cricoid cartilage had evidently sloughed out, and the ring was distorted. This gap was filled by a hyoid bone graft, and an acrylic mold inserted. In two months we were able to remove the tube perorally and decannulate the patient.

CASE 4.—I first examined this patient approximately one month after he had sustained severe crushing injuries to his right upper chest, cervical spine, and larynx. A tracheotomy had been done as an emergency, following the accident. The tube was apparently correctly placed, but there was no airway through the larynx. The cords were fixed in the midline. So we felt that we were dealing with a bilateral, recurrent nerve paralysis, in addition to a complete cicatricial stenosis. Because of the multiple injuries to the chest, cervical spine, and clavicle, nothing was done in regard to the larynx at this time.

Some six months after the injury, the laryngeal picture had not changed. So we made a midline incision in order to further investigate the injury and to insert a mold; however, we could find no evi-

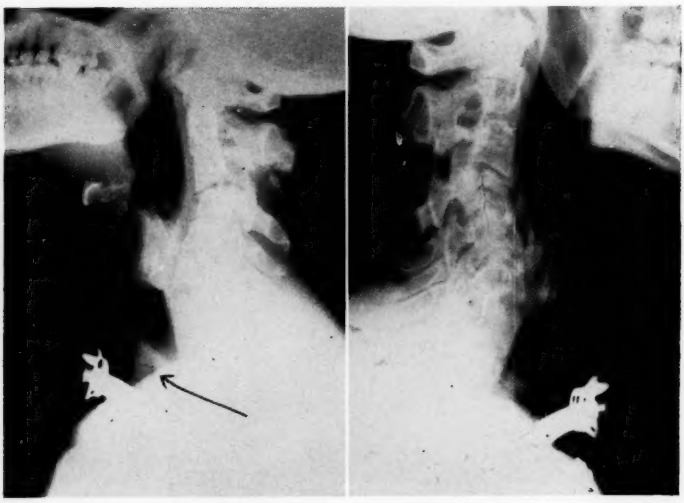

Fig. 4.—Preoperative lateral neck roentgenogram, showing marked distortion of cervical spine from multiple fractures. Arrow points to proximal end of evulsed trachea.

Fig. 5.—Postoperative lateral neck roentgenogram, after suture of trachea to cricoid ring and introduction of mold.

dence of the trachea after dividing the thyroid and cricoid cartilages. Dissection down the neck revealed the upper end at the level of the suprasternal notch. The tracheal cannula was in the lateral aspect, a few millimeters below the top ring. The trachea was dissected free, brought up to the cricoid by traction sutures, and resutured to the cricoid over a tantalum tube. However, we soon found that this tantalum tube was not well tolerated. Infected granulation tissue appeared which occluded the lumen; there was also considerable odor. Because of this, the tantalum tube was removed and a rubber tube inserted temporarily, but this likewise created more granulation tissue. So eventually everything was removed until the wound was well healed. A small airway was obtained but the lumen was no more than 3 millimeters. Because of the fractures of the cervical spine, it was almost impossible to do anything by direct laryngoscopy. Therefore, the larynx was reopened, the cicatricial tissue removed by sharp dissection, a mold inserted and its position maintained by

tantalum wire. A hyoid bone graft was placed with the hope not only of giving additional space in the area of stenosis, but also with the hope of obtaining an adequate airway between the paralyzed cords.

The patient was discharged on the sixth postoperative day, and has now gone five months in complete comfort. He is able to talk, is comfortable, and I believe the lumen will be adequate when the mold is removed within the next month.

XLVI

THE PRESENT STATUS AND THE FUTURE OF OTOLARYNGOLOGY

DEAN M. LIERLE, M.D.

IOWA CITY, IOWA

Is otolaryngology really in a dilemma? Has it reached a crossroads, uncertain of which way to turn?

For several years we have been hearing it referred to as a "dying" specialty; critics have declared that otolaryngologists did not distinguish themselves in the World Wars and were revealed to be poorly trained. Personally I feel that such comments, like the reports of Mark Twain's death, are greatly exaggerated. Dr. Lyman Richards summed up such comments in his excellent paper "The Dilemma of Otolaryngology", and his remarks prod us all into taking stock of our specialty and evaluating its place in the medical field.

With this in mind I have interviewed physicians, quizzed candidates taking the Board examinations, and considered the overlapping of other specialties with ours to discover whether current criticisms are valid and, if they are, to seek some remedy for the situation. Believe me, I have no occult power to look into the future but perhaps some of my conclusions will throw some light upon our problem. I hope my remarks will be interpreted as constructive criticism. Even though some of you may not agree with my conclusions, I shall be pleased if they will provoke some stimulating discussion.

Let me first give a brief review of a few events of the history of otolaryngology then present some opinions regarding its present status and finally hazard a guess as to what might improve the situation in the future.

Otolaryngology is one of the oldest specialties, and we have every reason to be proud of its history and accomplishments. According to history rhinology is perhaps the oldest of our specialties. Early

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kings were not immune to head colds, and it is said that the Egyptian Sekhet's emnuch in 3500 B.C. treated and healed the king's nostrils. Early laryngologists were physicians who found their interest in the chest extending to the area of the larynx. Early otologists were the surgeons.

The profession gained prominence in the thirties and forties of the nineteenth century, and France was the first country according to Stevenson and Guthrie to separate otology from the sphere of the surgeon and elevate it to a place of its own. Itard's text book, "Traite' des Maladies de l'Oreille et de l'Audition" was a landmark in the progress of our specialty. He did much to place otology on a sound scientific basis.

Desault of Paris is said to be the first to open the larynx by thyrotomy in 1810. In 1866 Patrick Heron Watson performed the first laryngectomy. Manoel Garcia, called the true father of laryngology, viewed his own glottis and trachea by holding a long-handled dental mirror against his uvula and deflecting rays of sunlight into his larynx.

The first special hospital for the eye and ear, Moorfields in London, was founded in 1805. In 1817 New London, Connecticut had an eye dispensary, and in 1820 Deerfield founded the New York Eye Infirmary which began treating diseases of the ear in 1822.

Fenton has called attention to the fact that for a generation after the Mexican war otolaryngology and ophthalmology "grew up" together. Eye, ear, nose, and throat specialists enlarged their knowledge through association with leaders in Boston, New York and Philadelphia . . . authorities who had studied in clinics in Paris, Vienna, and London.

These retrospective glances reveal that interest in our specialty goes back many, many years, but otolaryngology as we know it is a fairly new field. In the early days physicians were interested in the narrower areas of otology, rhinology and laryngology. The combined specialty of otorhinolaryngology was not practiced until the latter part of the 18th and the early part of the 19th centuries. It was not until that time, in my opinion, that the specialty was considered a science. Remember that the first hospital for the combined specialties of eye, ear, nose, and throat was established in 1822.

World War I revealed that education for the practice of ophthalmology and otolaryngology was not adequate. Consequently, the first examining board was organized in 1917 by the ophthalmologists. The board of otolaryngology was founded in 1924, seven years later. By that time the field of ophthalmology had broadened so that few ophthalmologists had any time for otolaryngology. Since the establishment of ophthalmology as a separate specialty, no one has encroached upon it because no one except ophthalmologists seem to know anything about the subject. As a result most ophthalmological services in the country today are autonomous.

Otolaryngology has made great advances in the last 10 to 15 years. This is particularly true in the field of otology. In this area we have observed fruitful research and an entirely new surgical technic. Tremendous interest has been aroused in the conservation of hearing. The study of the physiology in our field has been of great value and promises even greater fields of endeavor.

However, the time has come for serious review and adjustment. We hear that otolaryngology is a dying specialty—that everyone is encroaching on its field. There will always be an overlapping of interests in most specialties. New ones will always encroach upon the old. I do not believe ours is a dying specialty; I will concede that it has growing pains and that we are ready for certain adjustments.

What factors have been influencing otolaryngology, and what is its present status?

1. The board has attempted to raise standards. It has required the teaching of the basic sciences, has attempted to define the limits or scope of the practice, has set definite requirements for training candidates, and has devised comprehensive examinations. While the board's original function was evaluation of candidates, its scope has necessarily broadened over the years. It now assists in approving teaching institutions and cooperates with the institutions in stimulating interest in their undergraduate and graduate programs in the field of otolaryngology.

2. The advent of the newer drugs, chemotherapeutic agents and antibiotics, have left their imprint upon our specialty. Our field has definitely been narrowed by these drugs. The incidence of acute infections of the mastoid and sinuses with their complications has been markedly reduced. Whereas 20 or 25 years ago we would have

had in our service 150 to 200 cases of acute mastoiditis during the fall, winter, and spring seasons we now have only a few. Brain abscesses of otitic and sinus origin and infections of the lateral and cavernous sinuses are becoming rare.

3. The development of the specialty of chest surgery has restricted our field, and the end is not yet in sight. The chest surgeon has found it necessary to include the bronchoscope among his diagnostic tools. There is justification for this. One cannot deny that the chest surgeon should see the pathologic lesion that he later removes. The question arises as to who should do endoscopy. The otolaryngologist has always done these procedures and inasmuch as he is familiar with the diseases of the larynx and it is necessary for him to remove the foreign bodies, he feels he should continue to do the diagnostic procedures to maintain his technic. In addition to this there are not enough well trained chest surgeons who are capable of doing endoscopies to serve the entire country. The problem is being solved by either asking the chest surgeon to join the endoscopic team with the otolaryngologist or in sharing the material in the departments of chest surgery and otolaryngology. The first method, in my opinion, is superior in that it eliminates duplication of costly instruments and most important it avoids the necessity of two diagnostic procedures for the patient.

4. The general practitioner seems to be taking over a portion of the minor procedures in our field. This is particularly true in rural areas and smaller cities. I know of some places in the middle west where 80 per cent of the tonsillectomies are done by general practitioners. In most instances they are done well. The general practitioners also are doing myringotomies and treating acute rhinitis, acute tonsillitis, peritonsillar abscesses, and occasionally acute sinus disease. One cannot offer any objection if the particular physician is capable of doing the work. I have felt for a long time that the day is approaching when the general practitioner will be trained to do these simple procedures. Certainly the trend is toward more complete preparation in undergraduate teaching as well as in graduate training for the general practitioner. In some instances now their training includes otolaryngology.

A recent questionnaire sent to 574 general practitioners in Iowa showed that from 1½ to 80 per cent of their work was in the field of otolaryngology. The average was about 22 per cent from Sep-

tember to June. These reports were not planned studies but were estimates. I believe most of you would agree that it might be impractical for a general practitioner in a rural area to call on an otologist 20 miles away to do a myringotomy. In view of the fact that he is going to continue to do some of these procedures, I believe that the answer is better undergraduate and graduate training in otolaryngology for medical students and general practitioners.

A survey by a committee of the American Board of Otolaryngology with Dr. L. R. Schall as chairman concerned with undergraduate training in the medical schools of this country revealed increased interest in our field. Answers to the following questions were returned from forty-three institutions:

1. *Does your department teach anatomy of the ear, nose, and throat to medical students?* Affirmative answers came from 23 institutions, negative answers came from 13, and five said the teaching was done in review.

2. *In teaching physical diagnosis do you teach the technic of examination in otolaryngology?* There were 24 affirmative answers, the number of hours varying from two to 72.

3. *Do you give formal lectures?* 43 reported yes. The number of lectures ranged from six to 44.

4. *Do you give instruction by section in either outpatient department or the clinic?* The hours varied from eight to 373 in the outpatient department.

This information was very revealing, and Dr. Schall's committee recommended the following minimal requirements for undergraduate teaching in otolaryngology:

1. That the anatomy of the ear, nose, and throat be taught as part of the regular anatomy instruction in the freshman year.

2. That two hours of orientation and four hours of physical diagnosis be required in the sophomore year.

3. That forty hours of didactic and clinical instruction be included in the junior year, arranged according to the facilities available and at the discretion of the chairman of the department.

4. That elective courses be offered in the senior year.

Following World War II it was reported that the rank and file of otolaryngologists in the various military services obviously lacked proper training. Coming from some of you who were commanding officers, this criticism must have been justified. We know that in the army table of organization at that time the otolaryngologists were one grade lower than the ophthalmologists. This has been corrected, I understand. These men were trained as civilians; so no responsibility for their shortcomings is attributable to the military services.

I was determined to find the reason for this situation. For the last five or six years I have talked to all candidates taking board examinations regarding this seeming deficiency in training. From them I have obtained information regarding their training in the varous institutions. I have assured them that any specific information they gave me would be confidential, and this promise I intend to keep. The facts obtained were revealing and corroborated what I felt I already knew.

The most glaring deficiency in training seems to be the lack of uniformity in the scope or field of otolaryngology in our teaching institutions. The Board of Otolaryngology requires that candidates be examined in all phases of otolaryngology, peroral endoscopy, maxillo-facial surgery, and surgery of the neck exclusive of the thyroid gland. It has been revealed that only about half of the institutions teach the entire field of the specialty. In a number, the operative procedures consist merely of tonsillectomies and submucous operations. Some schools include endoscopic procedure, some plastic surgery of the ear, nose, and throat; and a very few, fenestration surgery.

Various reasons are given for the limited scope of training. One, often the most persistent, is that many departments are sections of general surgery and are not permitted to do neck surgery. I am reluctant to admit this because I am sure that in general we, ourselves, are responsible for this limitation. If we are capable no fair-minded general surgeon will object to sharing some of this work. The surgeon knows that if one is capable of treating primary malignancies of the ear, nose, and throat he must complete the job and resect the metastases in the neck when necessary. He knows that persistent thyroglossal ducts and cysts and branchogenic cysts are also in the field of otolaryngology. He will properly rebel against

turning any of these procedures to us if we are not capable of doing them well. We all realize that sharing this material is necessary for teaching in both specialties.

Other repeated criticisms concern lack of supervision, lack of organization of basic science courses, the complaint that most of the major operative procedures are done by the attending staff, and the lack of regard for the general condition of the patient.

During the last two years there have been fewer criticisms. Questionnaires reveal that 25 institutions out of 43 are now doing neck surgery; 26 are doing plastic surgery of the ear, nose, and throat; and 26 are doing endoscopic procedures.

I believe that the present status of our specialty is encouraging. The Board has made definite progress in raising standards. An encouraging number of institutions are adhering to these standards. A creditable job is being done in the teaching of the basic sciences. Not long ago the subject of pathology was a mystery to most candidates. Now they reveal at least a working knowledge of the subject. There is a general desire on the part of teaching institutions to include the entire field of otolaryngology in their programs. Some institutions are offering opportunities for research. I think the future of otolaryngology is in the hands of the country's teaching institutions. The general pattern is toward the development of more and better otolaryngologists.

What can the institutions do?

I. They can conform to the general conception of the scope of the field of otolaryngology. The scope, I believe, should adhere to the requirements as outlined by the Board. It is indicated in this statement: "This examination encompasses all phases of otolaryngology and peroral endoscopy, including maxillofacial surgery and surgery of the neck, excluding the thyroid gland."

II. Institutions must develop teachers capable of carrying out this program. Generally speaking, the method of selecting teachers has not been all that it should be. Training of young men who plan to become teachers should be broad. Remember that the excellence of the clinicians developed in our time was attributable to the fact that they had, for the most part, wide experience in general practice before training in the specialty. By the same token, our young teachers should have fundamental training in general surgery and

internal medicine, a minimum of two years of general surgery, one year of internal medicine and three years in otolaryngology.

III. Institutions should provide opportunities for research and should stimulate interest in this field for both staff and, to a more limited degree, house officers. There are many related fields, such as bacteriology, physiology, etc., in which investigations could be made by members of our profession.

IV. Good residency training is essential. Residency training requirements for formal training is at least three years for 16 of the 18 boards. The American Board of Otolaryngology will require three years of training after July 1, 1950. However, the board very wisely permits the candidate to take one year in general medicine, one year of general surgery, or one year of additional rotational internship in lieu of one of the three years' training in otolaryngology. It may be well to consider in the future giving credit for general practice experience. In addition, the candidate may be eligible to take the examination if he has completed four years in the combined training of ophthalmo-otolaryngology. Those who have restricted their practice for seven years and who have had training in the basic sciences may take the examination. All candidates of course must meet certain other requirements.

I have heard some criticisms regarding the long period of training required for otolaryngologists, particularly since the board has increased the requirements from two to three years. Some object to the extra cost and the additional time and declare that the extra year is not necessary. Some anticipate a dearth of residents. As a matter of fact, there are not enough residents for the training programs that have been approved at the present time. The reason for this is that since the war a large number of hospitals have been added to the list of approved residencies and fellowships. There has not been a decrease but a definite increase in the number of candidates for Board examinations. During the year of 1944, 159 candidates appeared for examination and in 1950 there will be 249 candidates examined. We still have a large number of applicants waiting for assignment to examination.

There are a number of reasons why a third year seems necessary. In the first place, few of the individuals going into the specialty have had general experience. They need the extra year for experience in general surgery, internal medicine, rotating internships or otolaryn-

gology. During a longer period of residency candidates will see and handle types of cases more rarely seen than in former years. During a three-year period a resident may familiarize himself with various operative procedures not available to him in a shorter residency. Again, most of the residents are married and have responsibilities away from their daily work, and the tempo, in general, is less rapid than formerly. Finally, the field is so much broader than it used to be that the additional time is necessary for adequate training. In our institution for the last ten years we have had training periods of from three to five years and have had no complaints regarding the time element involved.

There always has been a difference of opinion as to when the basic sciences should be taught. Some feel they should be introduced before the clinical work is begun. Others feel that they are better taught concurrently with the clinical subjects. We prefer a combination of the two methods. In our opinion, four months' study of anatomy, physiology, acoustics, pathology, principles of surgery, and didactic otolaryngology should precede the clinical training. We have found that there are frequently too many interruptions if these studies are given during the period of clinical training. Lectures in biochemistry, bacteriology, etc., may be given in seminar periods. Pathology should be continued during the entire period of training. We prefer to have the residents examine their own microscopic sections. These should be checked by staff members of the pathology department. An anatomical review, with a complete dissection of the head and neck, at the end of each year, will prove to be of definite value. Lectures and cadaver surgery for training in maxillofacial neck and sinus surgery may be offered in the evenings during the first and second years of residency. Journal, interdepartmental, and special seminars may be held daily between 1 and 2 p. m. Some of these periods may also be used for malignancy, cleft palate, and conservation of hearing clinics.

In addition to intensive training in the basic sciences during the first year time might be devoted to diagnostic procedures and minor surgery with the last two years given over to major surgery. Some attempt should be made to graduate the surgery. Supervision by staff members is necessary, of course, and it seems to me that residents should not attempt fenestration and maxillofacial surgery without additional special training. Those who wish to specialize in these

special divisions should have at least two years additional study, in my opinion.

V. Institutions should have adequate undergraduate programs. The amount of time for teaching varies greatly in different institutions. Replies to the questionnaire mentioned previously revealed a minimum of eight to a maximum of 373 clinical hours. We feel that physical diagnosis in otolaryngology should be correlated with that of internal medicine and a minimum of six to eight hours instruction be given during the sophomore year. A minimum of 32 didactic hours be available for the junior class, and a minimum of 72 hours in the outpatient department for seniors. I never could understand why every medical student shouldn't be taught to see a larynx as well as the big toe. Why physical diagnosis stopped at the clavicle I'll never know.

VI. Otolaryngologists should cooperate with the recently revived general practice program. Enough time should be allotted to otolaryngology to teach the practitioner the things he eventually must do in that field. The usual time allocated to otolaryngology is about two months. I believe the minimum should be at least four months. The general practitioner sees most patients with otolaryngological complaints first. Consequently he should have enough information about our field to manage certain cases and to determine which others he should refer to us for consultation.

VII. There has been considerable controversy during the last several years as to who should do plastic surgery in otolaryngology. Should it be done by the otolaryngologist or the general plastic surgeon? My answer is that it can be done by both. To me it is perfectly logical for an otolaryngologist, ophthalmologist, general surgeon, or urologist to do his own plastic surgery provided he has the ability and has been well trained in the field. I believe that this training should go beyond the usual residency, and I do not think every otolaryngologist can do plastic surgery. I would agree with Doctor Mosher in his statement that "The plastic surgeon must be a good dressmaker". There should be ample opportunity for men to receive training in plastic surgery in their own fields. One can understand the otolaryngologist's concern regarding the general plastic surgeon. The general plastic surgeon's field is broad and he is in a sense a rhinolaryngologist. In many instances he manages the malignancy of the ear, nose and throat; resects the metastases of the

neck when necessary; treats traumatic injuries including fractures and lacerations of the face; and does septal operations. Certainly the fenestration operation could be called a plastic procedure and could therefore be included in their scope. I wish to emphasize that I am definitely opposed to short inadequate training periods for otolaryngologists who expect to do plastic surgery.

VIII. Let's have a renewal of a teachers' section in the American Academy of Ophthalmology and Otolaryngology where ideas regarding the teaching of undergraduates and graduates and the promotion of research could be exchanged. Minimum standards could be determined through such exchange of ideas. Most of us know very little about each other so far as teaching is concerned.

Although in the far distant future, because of reasons outlined, the combined ophthalmologist and otolaryngologist may disappear from the picture, there is certainly a great demand for him at present, particularly in the rural areas. There are still 34 training centers in the United States for combined training. This combined training should be encouraged for the time being at least.

Otolaryngology may be in a dilemma but the future is not dark. It certainly is in no more serious straits than some of the other surgical specialties. The situation with us is improving and will continue to do so if the teaching centers will continue to fully accept their responsibilities. Overlapping of specialties is inevitable, and sharing of materials is necessary. Respect and confidence among the various specialties is essential. Sometimes specialism goes too far. There are times when it should make little difference who does which procedure. The question should be: Is it well done? I believe now is the time to reflect and re-chart our course. We must guard against a tendency to rest on the laurels of those vigorous pioneers who prepared the way for us. Let us guard against a feeling of complacency. And by all means, let us not blame others for our own shortcomings. If we attain excellence as specialists, we need not concern ourselves about what others do in our particular field.

XLVII

THE MODERN ROLE OF THE RHINOLOGIST IN THE DIAGNOSIS AND THERAPY OF THE ETIOLOGY OF HEAD PAIN

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My interest in the subject of head pain was first stimulated in 1929. In that year I attended a clinic conducted by Dr. Harvey Cushing. He referred in a sort of "off the record" comment to the meddlesome operations of the rhinologist in some cases of brain tumor which eventually came to his clinic. However, all of his comments concerning the otolaryngologist are not off the record. There is recorded in Kerrison's textbook in the chapter on Acoustic Neuroma an indictment by Dr. Cushing of the otologist, not for failing to make a diagnosis of this particular tumor, but for his failure to at least suspect one.

The term head pain is an inclusive reference which encompasses both headache and neuralgia. Stedman's medical dictionary defines headache as "a diffuse pain in various parts of the head not confined to the distribution of any nerve", and neuralgia as "a pain of a severe throbbing or stabbing character in the course of the distribution of a nerve".

Most of the literature on head pain has come from the investigations of neurologists, psychiatrists, and internists, though the rhinologist is often consulted for a clinical interpretation because the activities of his specialty are at a site where so much head pain is located.

You are undoubtedly aware of the fact that the literature on head pain is voluminous. In most instances when the literature on a medical subject is voluminous, there is considerable variation of opinion. In this instance there is no exception.

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It is not my purpose in this presentation to attempt a review of this literature. Rather, I would have the temerity to attempt to state in simple language just what we can apparently accept about head pain as factual and practical.

From the extensive investigations of recent years, three observations would seem to stand out as apparently of fundamental importance. These are:

1. The basic mechanism of the production of head pain in many instances is a stretching of the pain sensitive nervous structures along the blood vessels.

2. Autonomic dysfunction characterized principally by construction at the arteriolar level, which is followed by peripheral vasodilatation would seem to be an etiological factor in some instances of head pain.

3. A number of observers would accept the idea that due to physical or emotional stimuli a localized tissue damage may occur which results in a localized production of histamine and that the vasodilating effect of this substance produces pain.

On the basis of our knowledge to date, it would seem that head pain is produced in one of three possible ways:

1. By a change in cerebral blood volume. The greater cerebral arterial blood volume which occurs in the febrile state accompanying an infectious disease would be an example of this.

2. Direct irritation of a sensory nerve or nerves. The pain associated with dental disease or the headache produced by pressure from the swelling in acute maxillary sinusitis are examples of this mechanism.

3. The localized effect of histamine just referred to.

In a consideration of the problem of head pain in its entirety, one should as a clinician divide the subject of head pain into the categories of headache and neuralgia.

There are numerous classifications of headache. I first attempted one about ten years ago. It was based on Auerbach's classification. He divided the types of headache into three groups, one including the headaches due to general or systemic disorders, another those which seem more or less independent of any bodily disorder, and third, those which have their origin from some disorder localized within the head. I have found occasion to make changes

in this classification a number of times as the contributions of various observers have brought forth new ideas. I have found it useful to have a classification for two reasons; one is that it provides me with a more or less complete perspective when I am confronted with a patient whose problem is one of chronic head pain. The second reason is that it offers a useful example when we try to impress upon our students the importance of thinking of the relationship of general medicine to our specialty.

Today, I would offer this classification as follows:

Headache from Systemic or General Disorders

1. Infectious diseases or localized inflammations causing systemic disorder
2. Circulatory disturbances
3. Abnormal blood states
4. Gastro-intestinal disturbances
5. Emotional disorders, anxiety, or nervous states
6. Miscellaneous: in certain relaxation states, from caffeine withdrawal, etc.

Independent Forms of Headache, from disorders in

1. Muscles and fibrous structures about the head (Williams)
2. Vascular structures about the head (Williams)

Headache from Disorders Within the Head

1. Intracranial disease
2. Disorders of the eye
3. Nasal space disease

The time allotted for this discussion is so limited that I will omit reference to the headaches from systemic or general disorders except to remark that the components of this category emphasize the importance of studying the patient in his entirety particularly when there is a problem of differential diagnosis.

Williams has suggested a classification of certain types of headache grouped according to structures involved which I have placed in the so-called "independent forms", that is, those which are not part of a systemic or general disorder and are not part of a headache from a disorder actually arising within the head.

There probably will be no difference of opinion regarding the headache in the first part of the classification I have suggested, namely, those from systemic or general disorders. When one examines the next group he encounters the newer concepts which at this point are interesting to examine in some detail.

HEADACHE FROM MUSCLES AND FIBROUS STRUCTURES ABOUT THE HEAD

There are three causes of headache involving these structures: muscle tension, fibrositis, and myalgia. One has only to recall the anatomy of the muscular and fibrous structure about the head to realize that there are numerous possibilities for factors of tension, fatigue and inflammatory disturbance to operate at these sites.

Muscle tension is a common cause of headache. It may result from the maintenance of the head in certain positions sometimes occupational, tension due to anxiety, chronic fatigue, etc.

Fibrositis has been described as of two types, a primary and a secondary. The latter is very common. It is often associated with arthritic changes in the cervical spine. The headache is localized at the back of the head, particularly at the nuchal line. Chilling as occurs in cold damp weather often causes exacerbation. Mild exercise, physical therapy, and the salicylates improve the symptoms.

Primary fibrositis is said to be present when localized sensitiveness occurs in and around the scalp and head in which the pain tends to be of a bright burning character, with sensitiveness of the scalp to light manipulation.

Myalgia of the head is characterized by a headache. According to Williams it originates in muscle on the basis of a physical or intrinsic allergy. It is characterized by isolated, firm, tender areas in the body of certain muscles of the head and neck. These areas tend to recur in the same locations, and when the pain is present the areas are increasingly tender and the pain is referred in a myotomic distribution. The following muscles have been described as the site of myalgia: the upper border of the trapezius, the splenius capitis, the upper one third of the sternocleidomastoid, the temporal, the stylohyoid, the anterior belly of the digastric, the cricoarytenoideus posticus, the mylohyoid, the insertion of the glossopalatinus into the tongue, and the superior constrictor of the pharynx.

The sites of reference of pain are usually unilateral.

The sufferer from myalgia is usually extremely sensitive to drafts and atmospheric changes.

Williams suggests that the causation of myalgia is a spasmodic contraction of the arterial limb of capillaries resulting in a localized release of histamine with its vasodilating effect. The use of vasodilators such as nicotinic acid offer relief by their action in opening up the spasmodically contracted arterial limbs.

HEADACHE FROM VASCULAR STRUCTURES ABOUT THE HEAD

Since the basic mechanism of headache is often the stretching of pain sensitive structures, most head pain has something to do with blood vessels. There are three types of headache which according to modern knowledge are clinical entities and more or less set apart from the rest. These are: migraine, histaminic cephalgia and one that we have designated in the past as "sphenopalatine ganglion" neuralgia. The latter, according to modern concepts is probably a vasodilating head pain involving the internal maxillary artery.

Migraine. There seems to be an adequate basis for the hypothesis that migraine is due to uncompensated fluctuations of the effective arterial blood volume. An inherited familial defect allows the development of vascular spasm which is followed by an over dilatation of the extra cranial and intracranial arteries. The most effective treatment consists of the subcutaneous injection of ergotamine tartrate.

Histamine cephalgia is an uncommon type of headache occurring in the third decade or beyond, usually unilateral, periodic, of sudden onset and short duration. The mechanism of this form of headache has been explained on the basis of localized capillary constriction with release of histamine into the adventitia of the wall of the affected artery. Vasomotor rhinitis on the affected side, vasodilation in the skin, injection of the conjunctiva on the involved side and tearing are associated phenomena of autonomic overactivity. The treatment consists of giving vasodilators such as histamine or nicotinic acid although the administration at the height of the attack will temporarily increase the pain.

"Sphenopalatine ganglion neuralgia". Williams and Hilger both suggest that an explanation of this picture can be satisfactorily made

by considering it to be a vasodilating pain involving certain branches of the external carotid artery, principally the internal maxillary. I think most of you will agree that at least all previous explanations of this picture of headache were unsatisfactory. Fenton and Larsell, and Higbee, have pointed out that the actual structure of the sphenopalatine ganglion has no sensory nerve fibers although fibers from the maxillary division of the trigeminal nerve pass through it, and that transmission of pain by fibers of the autonomic nervous system can not be confirmed by any investigators. Dysart has suggested that the relief of pain when the sphenopalatine ganglion is anaesthetized might be explained on the basis that there is an interruption of a reflex arc in the sympathetic fibers in the ganglion. Hilger has recently stated that "cocainization of the sphenopalatine ganglion produces relief only if the spastic arteriolar bed is under control of the sympathetic fibers under blockage, or if sufficient intranasal vasoconstrictor is administered to give systemic absorption and direct constrictive influence on the dilated arterial trunk". Williams has reported relief of this socalled "sphenopalatine" pain in mild cases by the use of intramuscular injections of nicotinic acid and in the more severe cases by using the drug intravenously.

HEADACHE FROM DISORDERS WITHIN THE HEAD

The third major category in the classification of headache that I have suggested is concerned with the headaches that arise from intracranial disease, disorders of the eye, and nasal space disease. There is insufficient time to comment on intracranial disease except to remark that the headache from serious intracranial disorders such as neoplastic disease is still blamed on the sinuses in some instances particularly in the early stages of the disease, and that the neurosurgeon too often gets a history of a submucous resection or some operative sinus procedure being done to relieve a headache caused by a brain tumor, an aneurysm, etc.

A good ophthalmologist should experience no great difficulty in determining whether or not a headache is due to a refractive error, muscle imbalance, increased intraocular tension or an inflammatory condition in the eye.

Headache originating in the nasal space has two possibilities as to its causation. It may be caused by:

1. Contacts within the nasal fossae

2. Disease within a sinus causing pressure or tension.

The areas in which contacts may result in the production of pain are:

1. The nasofrontal area
2. The middle meatus
3. Between the septum and the lateral nasal wall.

The pain produced by these contacts is usually acute and rather temporary except that because of abnormal changes in the tissue, repetition of the pain may become frequent. In my experience I have never encountered any difficulty in definitely establishing the fact of whether or not a given pain was due to a congestive contact. This can be established by the simple process of cocainization and in some instances by simple vasoconstriction.

McAuliffe, Goodell and Wolff have demonstrated the pain sensitive areas in the nasal space and the regions to which pain originating from various portions of the nasal space is referred. When they stimulated the antero-inferior portion of the nasal septum, the lateral wall of the maxillary sinus, the middle and inferior turbinates, and the ostium of the maxillary sinus, the pain was usually referred to the malar and zygomatic regions of the homolateral side. Stimulation of the superior portion of the nasal fossa showed reference of pain to the eye, the infraorbital region and the junction of the nasal bridge and frontal bone. When the mucosa of the sphenoid was stimulated, a slight degree of pain was referred to the vertex of the skull.

In my experience, headache from tension or pressure within a sinus is common but disappears as the sinus drains or the swelling subsides. Headache which is a chronic problem is rarely encountered due to tension within a sinus, and when it does occur it is usually a matter of something unusual such as a mucocele or osteoma.

THE NEURALGIAS

Outside of the writings of Sluder and of Vail there has been very little offered by otolaryngologists on the subject of neuralgia and its role in the clinical problems of otolaryngology. Most of the contributions came from neurologists or neurosurgeons, and were characterized by detailed classifications indicating how little was actually understood about some types of headpain. Glaser in an

article in the Annals of Otology, Rhinology and Laryngology in 1939 suggested the classification of facial neuralgia in four main groups as follows:

1. Trigeminal neuralgia—primary and secondary
2. The otalgias
3. Other cranial nerve neuralgias
    a. cervical neuralgias
    b. facial (7th nerve) neuralgias
    c. glossopharyngeal (9th nerve) neuralgias
    d. vagus (10th nerve) neuralgia
4. Atypical neuralgia
    a. primary atypical neuralgia
    b. clinical entities manifesting atypical neuralgias:
        1. sphenopalatine or vidian neuralgia
        2. post-herpetic trigeminal neuralgia
        3. trigeminal ghosts
        4. trigeminal ghosts with lingual spasm
        5. mandibular joint pathology
        6. autonomic facio-cephalagia
        7. painful tic convulsive
        8. hypertonic neck muscles
        9. senile neuralgia
    c. systemic diseases producing atypical neuralgias:
        1. allergy
        2. endocrine disturbances
        3. psychoneuroses
    d. pathology in the head, chest and abdomen manifesting atypical neuralgia:
        1. infections about the head—
            (a) mastoiditis
            (b) cavernous and longitudinal sinus thrombosis
            (c) deep-seated facal abscesses
        2. tumors of the head and neck

3. intracranial pathology
4. dental pathology
5. nasal septum deviations and spurs
6. ocular pathology
7. chest pathology
8. abdominal pathology
9. pelvic pathology

I am inserting this classification only to remind you of the cluttered up state of thinking relative to this subject a few years ago.

Today, by making use of the modern concepts relative to the production of head pain, it seems possible to classify neuralgias in and about the head into a comparatively simple scheme as follows:

NEURALGIC HEAD PAIN

1. Trigeminal neuralgia, primary and secondary
2. Glossopharyngeal neuralgia, primary and secondary
3. From irritation of certain branches of
   a. the facial nerve (glossopalatine)
   b. the vagus nerve (auricular, superior laryngeal)
   c. the second and third cervical nerves (occipital nerves, greater auricular N.)

The diagnosis of primary forms of trigeminal neuralgia and of glossopharyngeal neuralgia can practically be made from a description of the symptoms since both are such clear cut clinical entities. There should be little difficulty in diagnosing the "Tic Doloureaux" of these nerves.

Secondary forms of trigeminal neuralgia are encountered practically every day in the practice of otolaryngology. The many causes include dental origins, inflammations, vascular lesions, tumors, trauma, from such diseases as multiple sclerosis, syphilis, etc.

The secondary form of glossopharyngeal neuralgia is most frequently encountered as the aftermath of tonsillectomy in the immediate postoperative period.

Neuralgic disturbances from irritation of certain branches of the facial nerve are uncommon and apparently are limited to inflam-

mations involving the geniculate ganglion. The pain is projected in an area limited to a portion of the tragus, concha, external auditory canal and tympanic membrane. Neuralgic disturbances in the form of earache which occurs through the superior laryngeal branch and auricular branch of the vagus and its sensory branches related to the pharyngeal plexus are not uncommon from malignant lesions on the rim of the larynx, involving the epiglottis or the pharynx.

Neuralgic disturbances involving branches of the second and third cervical nerves are invariably the result of local inflammation, trauma, etc.

SUMMARY

The voluminous literature concerning the subject of head pain as it occurs in the form of headache and neuralgia has been contributed largely by neurologists, psychiatrists and internists despite the fact that in the every day practice of clinical medicine, it is the otolaryngologist who is often consulted for diagnosis and treatment.

The advances in our knowledge of the etiology of head pain involve three considerations:

1. The basic mechanism of the production of head pain is in many instances a stretching of the pain sensitive nervous structures along the blood vessels.

2. Autonomic dysfunction is a major factor in the production of some forms of head pain.

3. The vasodilating action of histamine released locally in certain forms of tissue change as may occur from physical stimuli and autonomic dysfunction may explain the mechanism by which vasodilation occurs.

On the basis of this newer knowledge, a simplified classification of headache and of neuralgic head pain is suggested.

REFERENCES

1. Auerbach, S.: Headache; Its Varieties, Their Nature, Recognition and Treatment; a Theorectical and Practical Treatise for Students and Practitioners, London, Oxford University Press, 208 pp. 1913.

2. Dysart, B. R.: Modern View of Neuralgia Referrable to Meckel's Ganglion; Report of Cases Showing Relief of Pain and Sometimes Arrest of Development of Ulcers of the Cornea by Cocainization of the Ganglion, Arch. Otolaryng. 40:29-32 (July) 1944.

3. Higbee, D.: Functional and Anatomic Relation of Sphenopalatine Ganglion to Autonomic Nervous System, Arch. Otolaryng. 50:45-58 (July) 1949.

4. Hilger, J. A.: Carotid Pain, Laryngoscope 59:829-837 (August) 1949.

5. Glaser, M. A.: The Facial Neuralgias: Their Etiology and Treatment, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 48:324-345 (June) 1939.

6. Horton, B. T., MacLean, A. R., and Craig, W. McK.: A New Syndrome of Vascular Headache; Results of Treatment with Histamine; Preliminary Report, Proc. Staff Meet., Mayo Clinic 14:257-260 (April 26) 1939.

7. Horton, B. T.: Headache: Clinical Varieties and Therapeutic Suggestions, M. Clinic North American (Mayo Clinic Number) 973-1005 (July) 1949.

8. Larsell, O., and Fenton, R.: Sympathetic Innervation of the Nose: Research Report, Arch. Otolaryng. 24:687-695 (Dec.) 1936.

9. McAuliffe, G. W., Goodell, H., and Wolff, H. G.: Experimental Studies on Headache: Pain from Nasal and Paranasal Structures, A. Research Nerv. and Ment. Dis. Proc. 23:185-208, 1943.

10. Moensch, I. G.: Headache, Yearbook Publishers, Inc., Chicago, 1947.

11. Muller, O.: Ueber den Saftestrom in Blut Gewebsspalten und Lymphbabnen, Schweiz Med. Wchnschr 70:365-369 (April 27); 617-621 (June 29) 1940.

12. Selye, H.: The General Adaptation Syndrome and the Diseases of Adaptation, J. Clin. Endocrinol. 6:117-230 (Feb.) 1946.

13. Sluder, G.: Headache and Eye Disorders of Nasal Origin, St. Louis, C. V. Mosby Co., 1940.

14. Ray, B. S., and Wolff, H. G.: Experimental Studies on Headache; Pain Sensitive Structures of the Head and Their Significance in Headache, Arch. Surg. 40:813-856 (Oct.) 1940.

15. Vail, H. H.: Neuralgia of the Head and Face Distinct from Trigeminal Neuralgia, Pract. Lib. Med. and Surg., D. Appleton Century Co. 11:891-902, 1937.

16. Williams, H. L.: The Syndrome of Physical or Intrinsic Allergy of the Head; Myalgia of the Head (Sinus Headache), Proc. Staff Meetings of the Mayo Clinic 20:177-208 (June 13) 1945.

17. Williams, H. L.: Differentiation, Cause and Treatment of Certain Common Types of Headache, Graduate Lecture, Amer. Acad. Ophth. Otolaryng. 1947.

18. Wolff, H. G.: Headache and Other Head Pain, New York, Oxford University Press, 1948.

XLVIII

EFFECT OF CORTISONE ON IDIOPATHIC GRANULOMA OF THE MIDLINE TISSUES OF THE FACE

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At the meeting of the American Laryngological Association last year, one of us (H. L. W.) presented a review of the literature on lethal granuloma of the midline facial tissues together with reports of three cases.[1] Pathologic study of these cases and a review of similar cases indicated that idiopathic facial granulomas frequently were not localized disorders but that, in addition to the destructive facial lesions, diffuse disease of the arterial system and peripheral capillary bed was present.[1-7] These lesions consisted of periarteritis nodosa, necrotizing arteritis, granulomatous formation and the development in one case of lupus erythematosus disseminata. Review of the literature revealed convincing evidence that a stereotyped reaction of the peripheral vascular bed, consisting of arteriolar spasm with dilatation of the capillary and venule, was the underlying lesion in all inflammation.[8-32]

Extensive pathophysiologic investigation has shown that this type of reaction is common to inflammation, in the widest sense of the word, which includes the allergies and the so-called collagen diseases.[33-35] Ricker[8] has presented evidence that such a reaction cannot occur in the absence of autonomic nerves. In other words, this stereotyped vascular reaction is a fundamental part of the physiologic mechanism by which the body resists stress in its external or internal environment, and by which it tends to restore physiologic

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equilibrium or homeostasis.[36-39] The reaction occurs whether the stress is produced by atmospheric changes or changes in temperature, by the invasion of micro-organisms, by the injection of nontoxic protein material, or by changes in the chemical or hormonal composition of the tissue fluids.

This physiologic mechanism known as the "autonomic system"[36] is made up of three functionally inseparable parts: (1) the semipermeable cell membranes and the tissue fluids in which the physicochemical reactions take place; (2) the hormonal system by which such reactions are speeded, and (3) the autonomic nervous system by which such reactions are localized and directed. Any change in the function and activity of one part of this triad immediately involves modifications in the other two, thus presenting a synchronized complex action of the entire system.[40] Variation in the nutritive demands of the tissues results from changes in environmental conditions, and the reactions of the autonomic system to them. The purpose of the capillary bed is to convey nutrition to the tissues and to carry away waste material. Therefore the capillary bed is of necessity involved in any reaction of the autonomic system. Normal variations in the tonus of the blood vessels occur simultaneously in the whole body or in a large part of the body and nutrition of the tissues is maintained according to environmental demand.

An abnormal type of reaction of the vascular bed to stimuli from the autonomic system is spasm. The most important thing about spasm is that it may be limited to a certain region, organ or part of the body or even to one artery. Spasm in this sense is always a localized phenomenon, a pathologic occurrence.[40]

Ricker[8] demonstrated that a normal animal reacted to severe injury with arteriolar spasm and capillary dilatation. It is known that certain persons react maximally in this manner to stimuli which in the case of the normal person would produce a mild reaction; a tendency develops to react to stimuli to which a normal person would not react, and they react to degrees of stimulation which would not provoke reaction among normal persons.[41] This hyperactivity appears to be inherited as a constitutional tendency which depends on an inherited lability of the autonomic system and is known as "autonomic dysfunction" or "allergy." The reaction is limited to a region, a tissue or an organ.[42] Arteriolar spasm and capillary dilatation compose the fundamental reactions common to all types of auto-

nomic dysfunction. The result is sludging of the blood and localized anoxia[43, 44] followed by injury to the cells and the release of such substances as histamine, heparin, necrosin, and leukotaxine depending on the type of cell injured.[45] An antigen-antibody mechanism may or may not be present. When present it is a secondary phenomenon.[43, 46]

The arteriolar and capillary reactions which should be stressed are functional disorders and can be observed only by means of biomicroscopy.[16] Possibly by virtue of the anoxic conditions produced or possibly by enzymatic activation (hyaluronidase)[47] they may lead to organic change.[48] The earliest organic change observed is fibrous degeneration of the collagen in the walls of the capillaries and in the media of the arterial walls of the larger vessels.[49] It was because of this reaction of collagen that Klinge[50] first termed these disorders the "collagen" diseases.[50-52]

The hormones of the anterior pituitary-adrenal cortex are known to control electrolyte balance, carbohydrate metabolism and capillary permeability;[53-63] they seem[64] to control the production and transportation of antibodies and might well be entitled the "master hormones" of autonomic function or homeostasis. It would seem reasonable to suppose, therefore, that some disorder of the mechanism which stimulates the anterior pituitary gland, or some disorder of the anterior pituitary gland itself or of the adrenal cortex might be the background for autonomic dysfunction.[65] Since evidence suggests that idiopathic granuloma of the midline facial tissues is an instance of autonomic dysfunction,[1-3, 6] it would seem reasonable to suppose that the use of cortisone in the treatment of this condition might produce a favorable effect. This is especially so in view of the fact that related disorders such as lupus erythematosus disseminata,[66] periarteritis nodosa,[67] asthma and vasomotor coryza[68] have responded favorably to such therapy.

In a case in which the diagnosis was idiopathic granuloma of the midline tissues of the face, we decided to try the effect of injections of cortisone. The rarity of the disease, the observation of the patient from the beginning of the disease through its long course and the effects of cortisone on a lesion of this type warrant our presentation of the following case.

REPORT OF CASE

A white man, 48 years of age, was first seen by one of us in 1943 and gave the following history: At the age of 38 years he had all his upper teeth extracted because of extensive pyorrhea. A denture which he wore from that time on caused him considerable discomfort in spite of repeated adjustments. In 1941 a small ulcerated area was noticed on the alveolar ridge on the left side and the attending dentist considered the ill-fitting denture to be the cause. A new denture and various local applications of antiseptics, however, did not influence the ulcerative process. Within one and a half years it had eroded through the alveolar process into the cavity of the maxillary sinus. Several attempts by an oral surgeon to close this fistula failed. At the time the patient was first seen the outstanding findings were a large antral-alveolar fistula and profuse purulent discharge through the fistula and from the left nasal cavity. Roentgenograms showed diffuse density of the entire left maxillary cavity which was felt to be consistent with the presence of chronic suppurative sinusitis. A Caldwell-Luc operation was performed; the antral cavity was found to be filled with purulent material and granulation tissue. Destruction of bone involving the anterior maxillary wall and floor of the antrum was discovered. The necrotic tissue was completely removed and an attempt made to close the antral-alveolar fistula. The pathologist reported the presence of granulation tissue lined with stratified columnar epithelium, a diffusely edematous corium, active proliferation of fibroblasts, new capillaries, diffuse infiltration of many plasma cells and neutrophils and focal collections of lymphocytes. The diagnosis was that of chronic suppurative inflammation of the antrum.

It soon became evident that the destruction of bone had not been arrested; further absorption of the alveolar process and floor of the antrum took place and a large oral antral-palatal fistula developed. Extensive exenteration of all the involved soft tissue and necrotic bone was done on several occasions in the next six months.

From June, 1943, until May, 1948, the patient felt fairly well; he gained weight; he was able to work steadily, and it appeared that the destructive process was arrested. A dental prosthesis was made to occlude the antral-palatal fistula.

In 1948 the patient began to have considerable pain in the malar-parietal region; the discharge from the antral cavity increased and a slight proptosis of the left eye became noticeable. Roentgenologic findings revealed evidence of what appeared to be complete destruction of the malar bone, zygomatic arch and frontal process of the malar bone, and uniform, increased density of the entire maxillary sinus on the left side. The lesion appeared to extend posteriorly and involved the greater wing of the sphenoid. The roentgenologists considered the entire process as unusual and not suggestive of ordinary bacterial osteomyelitis. They suggested an unusual granulomatous type of infection or a low-grade tumor, such as fibrous dysplasia of bone, as possibilities. A slight amount of osteitis was taking place in the wing of the sphenoid, and it was concluded that before it was destroyed the malar bone itself had been slightly denser than the normal malar bone. Sequestra were not present.

In July, 1948, a further attempt was made to arrest the destructive process. The external carotid artery was ligated and again extensive exenteration of the malar bone was performed and portions of the maxilla and pterygoid process of the left sphenoid were removed. The histopathologic findings included small pieces of highly vascular, inflammatory granulation tissue covered by a layer of epidermis

which penetrated deep into the granulations but did not show the invasive character of a tumor. The granulation tissue showed active proliferation of fibroblasts, new capillaries and diffuse infiltration of many plasma cells and neutrophils. The lining epithelium formed small, cystic spaces in the substance of the granuloma, and these were filled with pus. Fibrotic, necrotic and hemorrhagic areas were scattered through the granuloma. The only micro-organisms recognized on staining with hematoxylin and eosin were of the genus *Monilia*. The diagnosis was chronic inflammatory granulation tissue.

Numerous bacteriologic, serologic and hematologic examinations did not reveal any specific etiologic factor.

Again the condition appeared to improve for several months, but as before it was soon evident that the destructive process was advancing. During the year of 1948 the patient received extensive chemotherapy and antibiotic therapy, but neither one influenced the course of the disease in the least. In November, 1948, the patient was referred to the Mayo Clinic. He was 5 ft. 10 in. tall (177.8 cm) and appeared emaciated; he weighed 103 lb. (46.7 kg), although he stated that his normal weight was 128 lb. (58.1 kg.) The lesion in the left malar area, nose and palate was as described. Clinically the lesion appeared to be a typical midline idiopathic granuloma. Consultants in clinical bacteriology and mycology were called and they felt that if sufficient care and time were taken for bacteriologic and mycologic study, a causative micro-organism would be found. They felt, however, that in view of the nature of the lesion a gram-positive organism could hardly have produced it. The use of penicillin was advised to eliminate such organisms so that they would not interfere with the bacteriologic identification of some more likely bacterium. Accordingly 600,000 Oxford units of penicillin in oil was injected intramuscularly daily. Results of the Kline, Kahn, Hinton and Kolmer tests for syphilis were reported to be negative. A roentgenogram of the thorax revealed evidence of emphysema. Examination of the blood showed 4,430,000 erythrocytes and 11,900 leukocytes per cubic millimeter; 12.4 mg of hemoglobin per 100 cc of blood; a differential white count of 90.5 neutrophils, 4.5 of each lymphocytes and monocytes and 0.5 basophils per 100 cc and no eosinophils. A blood smear showed mildly toxic changes in the polymorphonuclear leukocytes. Urinalysis did not reveal any abnormality. The excretion of 17-ketosteroids in the urine was reported as 6.6 mg in 24 hours.

Examination of the eyes showed proptosis of the left eye. The exophthalmometric reading at 94 mm was right eye 18, left 25.

The result of the tuberculin test performed with purified protein derivative, single strength, was reported to be positive+++. Skin tests with histoplasmin and coccidioidin gave negative results. Repeated tests of sputum failed to reveal acid-fast organisms. Repeated smears of exudate from the granulomatous lesion in the antrum were negative for acid-fast bacilli.

After preparation with penicillin was felt to be complete, the patient was anesthetized and extensive specimens were taken from the interior of the left antrum for biopsy. In addition, four small lymph nodes were removed from the upper cervical region. A part of the tissue removed was sent for bacteriologic study, the rest being sent for pathologic examination. The pathologic report on the tissue removed from the antrum was granulomatous inflammation. The lymph node was reported to show inflammation with occasional eosinophils and moderate proliferation of endothelial cells. No organisms were seen.

On culture of tissue from the left antrum the following micro-organisms were found: (1) streptococcus (green-producing), (2) *Hemophilus influenzae* (many), (3) *Streptococcus hemolyticus* (few), (4) *Aerobacter aerogenes*, (5) *Neisseria catarrhalis* and (6) diphtheroids. Fungi and *Brucella* were not present. Cultures were negative for fungi and cultures for tuberculosis were reported negative for acid-fast bacilli. Twelve guinea pigs were inoculated with material from the antral granuloma. Three died and the examination was considered a failure. The remaining animals were killed after two months and reported negative for *Mycobacterium tuberculosis.*

In March, 1949, the patient was readmitted for further study. Three consecutive stools were secured and cultured for *Histoplasma capsulatum* with negative results. Repeated smears of the exudate from the antrum were reported negative for acid-fast bacilli. Sternal puncture was done and reported to show normal structure. Erythropoiesis appeared to be adequate and nothing was seen which would aid in diagnosis. Blood counts continued to show mild toxic changes with a relative increase of polymorphonuclear leukocytes. Roentgenograms of the head showed a destructive lesion of the left maxilla and frontal process of the left malar bone.

The patient was a heavy salt eater, and Selye[65] suggested that excess of the sodium ion with a relative alkalinity of the tissue fluids favored the development of necrotic vascular lesions. The patient was placed on a diet containing 0.5 gm of sodium, and given 6 gm of ammonium chloride daily, in divided doses. Since avitaminosis had been suggested as a causative factor in such lesions,[32] 100 mg of ascorbic acid for its possible effect on the adrenal cortex and 1 ampule Solu-B for its lipotropic effect and supposed effect on the blood vessels were given daily. He rebelled at this treatment after two weeks. No observable improvement had taken place.

The patient was readmitted in September, 1949, for the purpose of trying treatment with compound E acetate or cortisone. He appeared extremely emaciated and tired, his appetite was poor and he had an extreme distaste for meat.

On examination a thick, white discharge could be seen on the floor of the nose on the left. The naso-antral window was blocked by granulation tissue. On looking into the mouth it could be seen that the bone of the alveolar process on the left was deficient, this structure being made up of soft tissue. The gingivo-buccal fold on the left was absent and a large oro-antral fistula took its place. In the midline of the hard palate there was a perforation about 2 cm in diameter into the left antrum just lateral to the inferior turbinate. Granulation tissue protruded through this fistula and the antrum was seen to be almost entirely filled with a granuloma covered with a thick tenacious exudate, which could be removed only with difficulty, leaving a surface of unhealthy looking granulation tissue from which blood oozed. A rust colored paste-like exudate lay on the floor of the antrum. On palpation the lower rim of the left orbit appeared to be deficient. The structure of the malar bone beneath the tissues of the left cheek could not be determined, but a firm, rather resilient, mass could be felt extending laterally and posteriorly almost to the temporomandibular joint up into the temporal region and into the left orbit. The impression was obtained that the granuloma in the antrum had extended into the orbit and laterally into the cheek, displacing the eye forward and destroying the malar bone. This area was exquisitely tender to palpation.

A roentgenogram of the head taken at this time was reported to reveal evidence of irregular destruction of the left antrum. The floor and lateral wall of the orbit had been destroyed since the roentgenograms taken six months before, and there appeared to be an area of destruction in the roof of the orbit. The destruction of the zygomatic process of the frontal bone had increased also since the last roentgenogram.

The patient was seen again by a consultant in clinical bacteriology and mycology who stated that in spite of the fact that previous studies had failed to reveal any specific microbiologic agent, more studies should be undertaken because the granuloma was increasing in size. He drew attention to the fact that the patient's father had died of tuberculosis and that positive results for the tuberculin test had been obtained. He suggested that the granuloma in the orbit be exposed and material for study be secured from this area. We hesitated to do this owing to the introduction of an uncontrolled variable into the trial of the effect of cortisone on the lesion in the malar area.

Repeated cultures and smears of sputum were reported negative for acid-fast bacilli. Four guinea pigs were inoculated; at the end of two months the animals were killed and evidence of tuberculosis was not found in any instance.

Examination of the eyes at this time showed an exophthalmometric reading at 95 mm of 16 for the right eye and 26 for the left eye. Injection of the palpebral conjunctiva of the left eye was associated with several small fistulae of the skin of the outer canthus of the left eye. The subcutaneous tissue in this area felt indurated and was tender.

An initial dose of 200 mg of cortisone was given intramuscularly on September 17, 1949. Almost immediately the patient had a feeling of well-being; his appetite improved, and the tenderness in the left malar region practically disappeared. The granulomatous surface in the antrum began to smooth out; the antral discharge became thinner, and the surface exudate less dense. Improvement continued for about a week, reached a standstill and then there seemed to be a relapse in the general condition. The tenderness in the left malar region returned. No further change appeared to take place in the condition of the granuloma. At the end of four weeks of treatment the patient complained of sudden weakness and his weight dropped 6 lb. (2.7 kg) in two days. Blood sodium and the carbon dioxide combining power were found to be normal, chlorides were just at the lower limit of normal, but potassium was definitely low, showing 12.6 mg (3.2 mEq) per 100 cc of serum. Administration of cortisone was stopped and the patient was placed on a high potassium diet supplemented by potassium chloride. As soon as the administration of cortisone was stopped the patient's appetite decreased considerably.

Treatment was resumed after two weeks. The patient's general condition again improved and his feeling of well-being and his appetite returned; the changes were not so marked as at first, however, nor did they last so long. The granuloma in the antrum, however, continued to show steady improvement. The cavity of the antrum began to enlarge with the disappearance of the granuloma; laterally a considerable cavitation appeared where the lateral bony wall of the antrum had been destroyed. The exudate over the surface of the granulations disappeared almost entirely and healthy granulation tissue appeared. The firm area in the zygomatic region began to soften and tenderness disappeared to the point that considerable pressure could be exerted, although pressure temporarily increased the proptosis of the left eye to a marked degree. It was felt that more regression

had taken place in the granuloma in ten days than had taken place in the entire previous period of improvement. The improvement, however, then slowed down and nearly reached a standstill.

Owing to the continued skepticism of the consultant on clinical bacteriology as to the diagnosis of idiopathic granuloma, it was decided to open the fluctuant mass in the left orbit. About 120 cc of liquefied necrotic material was evacuated and sent for bacteriologic study. Mild dyspnea developed which was felt to be on the basis of a spontaneous pneumothorax. A mild degree of general weakness followed.

It seemed that a long period of treatment with cortisone would be necessary to clear up this lesion completely since improvement was not continuous but tended to diminish after a week of therapy. It was felt that treatment with cortisone (compound E) had been adequate to demonstrate that the granuloma was favorably affected, and it also appeared that further therapy might turn into a race between improvement in the granuloma and deterioration of the patient's general condition.

Cultures, smears and inoculations of guinea pigs from the necrotic material in the left orbit showed no evidence of tuberculosis.

Since the patient has returned home his condition has remained stationary.

COMMENT

In spite of attempts to identify a possible etiologic agent by repeated animal inoculation and cultures for bacteria, yeasts, fungi and viruses the consultant in bacteriology felt the condition might still be of microbial origin.

The nonspecific nature of the lesion, however, has been as well demonstrated bacteriologically as any of the lesions reported in the literature as idiopathic granuloma, and clinically, the lesion fits the classification of idiopathic granuloma of the midline facial tissues.

If either the infectious or allergic hypothesis is accepted for this condition, however, any observations as to the effect of cortisone would be equally valid, for many independent studies have indicated that unless specific organisms are found in the lesion, idiopathic and infectious granulomas cannot be distinguished. It would seem justifiable to assume that the same physiologic mechanism of resistance is present in both types.

In our case cortisone appeared to confer a brief temporary feeling of well-being and improvement in appetite. The reason for this effect is not known.

Early in the treatment with cortisone the patient was freed of pain and tenderness, but these symptoms returned in a short time.

The improvement of the patient and recurrence of symptoms could not be associated with any change observed in the local lesion. The possibility that these might be central effects was considered.

As soon as therapy with cortisone was started there appeared to be a definite and progressive change in the granuloma. The secretions became somewhat more profuse and thinner. The granulomatous tissue that could be seen slowly receded, the thick tenacious exudate slowly disappeared from its surface and the granulations became slightly firmer and of brighter color. The somewhat woody induration of the tissues of the left malar region found on palpation disappeared, the tissues became softer and those in the orbit seemed fluctuant. As it is well known that granulomas may heal by liquefaction necrosis, it was felt that the changes observed were the result of this process. These changes, however, could have been brought about by improved nutrition resulting from a more normal blood supply or by antigenic material (if the condition was felt to be due to invasion of micro-organisms) being brought to the area and liberated by the lymphocytes.

For a short period the patient suffered from exquisite tenderness of the left temporal artery. This suggested a vascular effect. We have no adequate explanation for the relatively rapid improvement in the granuloma on the first administration of cortisone or for the gradual slowing of the rate of improvement after a short time. The hypothesis which we presented that idiopathic granuloma had a stereotyped vascular defense mechanism in common with the resistance mechanism of the body could not be confirmed or denied by our study. However, the improvement in the local lesion supports the formulation that hormones of the adrenal cortex play some part in the physiologic mechanism of immunity.

Although cortisone had a healing effect on the granuloma, the progress was so slow as to make it seem that cortisone will not prove to be a useful drug in treatment for this condition and those of similar nature. This seemed particularly evident in view of the unfavorable side effects produced; namely, interference with normal metabolism of potassium and production of some general deterioration in the condition of the patient.

MAYO CLINIC.

REFERENCES

1. Williams, H. L.: Lethal Granulomatous Ulceration Involving the Midline Facial Tissues, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 58:1013-1054 (Dec.) 1949.

2. Wegener, F.: Über eine eigenartige rhinogene Granulomatose mit besonderer Beteiligung des Arterien-systems und der Nieren, Beitr. z. path. Anat. u. z. allg. Path. 102:36-68 (Jan.) 1939.

3. Weinberg, Tobias: Periarteritis Nodosa in Granuloma of Unknown Etiology, Am. J. Clin. Path. (Tech. suppl.) 16:784-791 (Dec.) 1946.

4. Boyd, William: Discussion, Am. J. Path. 22:646 (May) 1946.

5. Duff, G. L.: Discussion, Am. J. Path. 22:646 (May) 1946.

6. Rich, A. R.: Hypersensitivity in Disease; with Especial Reference to Periarteritis Nodosa, Rheumatic Fever, Disseminated Lupus Erythematosus and Rheumatoid Arthritis, Harvey Lect. s. 42, 1946-1947, pp. 106-147.

7. Sweeney, A. R., Jr., and Baggenstoss, A. H.: Pulmonary Lesions in Periarteritis Nodosa, Proc. Staff Meet., Mayo Clin. 24:35-43 (Jan.) 1949.

8. Ricker, G.: Die Methode der direkten Beobachtung der lokalen Kreislaufsstörungen und die Verwertung pathologisch-anatomischer Befunde in dem Kreislaufsorganen für die Pathologie derselben. In: Abderhalden, Emil: Handbuch der biologischen Arbeitsmethoden, Abt. VIII, Teil 1, (Erste Hälfte), Berlin, Urban & Schwarzenberg, 1924, pp. 509-560.

9. Oertel, Horst: Outlines of Pathology; in Its Historical, Philosophical, and Scientific Foundations; a Guide for Students and Practitioners of Medicine, Montreal, Renouf Publishing Co., 1927, 487 pp.

10. Wittmaack, Karl: Über die pathologisch-anatomischen und pathologisch-physiologischen Grundlagen der nichteitrigen Erkrankungsprozesse des inneren Ohres und des Hörnerven, Arch. f. Ohrenh. 99:71-136, 1916.

11. Lewis, Thomas, and Landis, E. M.: Observations upon the Vascular Mechanism in Acrocyanosis, Heart 15:229-246 (Dec.) 1930.

12. Abell, R. G., and Schenck, H. P.: Microscopic Observations on the Behavior of Living Blood Vessels of the Rabbit During the Reaction of Anaphylaxia, J. Immunol. 34:195-213 (Mar.) 1938.

13. Parrisius, Walter: Kapillarstudien bei Vasoneurosen, Deutsch. Ztschr. f. Nervenh. 72:310-358 (Oct.) 1921.

14. Kistiakovsky, E. V.: Erythrocyanosis Cutis Symmetrica; Angioneurosis Endocrinopathica Polyglandularis, Arch. Dermat. and Syph. 20:780-796 (Dec.) 1929.

15. Brown, G. E.: The Skin Capillaries in Raynaud's Disease, Arch. Int. Med. 35:56-73 (Jan.) 1925.

16. Lombard, W. P.: The Blood Pressure in the Arterioles, Capillaries, and Small Veins of the Human Skin, Am. J. Physiol. 29:335-362 (Jan. 1) 1912.

17. Ellis, L. B., and Weiss, Soma: The Measurement of Capillary Pressure under Natural Conditions and after Arteriolar Dilatation; in Normal Subjects and in Patients with Arterial Hypertension and with Arteriosclerosis, J. Clin. Investigation 8:47-67 (Dec.) 1929.

18. Parrisius, W.: Anomalien des periphersten Gefässsystems als Krankheitsursache speziell bei Menière und Glaukom, München. med. Wchnschr. 71 (pt. 1):224-225 (Feb. 22) 1924.

19. Mygind, S. H., and Dederding, Dida: Studies on Some Cutaneous and Subcutaneous Phenomena and Their Relation to the Labyrinthal Alterations in Mb. Menièri, Acta Oto-laryng. 13:474-486, 1929.

20. Mygind, S. H., and Dederding, Dida: The Significance of Water Metabolism in General Pathology as Demonstrated by Experiments on the Ear, Acta Oto-laryng. 17:424-468, 1932.

21. Redisch, Walter, and Pelzer, R. H.: Capillary Studies in Migraine; Effect of Ergotamine Tartrate and Water Diuresis, Am. Heart J.: 26:598-609 (Nov.) 1943.

22. Ricker, G., and Regendanz, P.: Beiträge zur Kenntnis der örtlichen Kreislaufsstörungen. Nach Untersuchungen am Pankreas und seinem Bauchfell, an der Conjunctiva und dem Ohrlöffel des Kaninchens, Virchows Arch. f. path. Anat. 231:1-184, 1921.

23. Fremont-Smith, Frank, Morrison, L. R., and Makepeace, Alexander: Capillary Blood Flow in Man During Fever, (Abstr.) J. Clin. Investigation 7:489-490 (Aug.) 1929.

24. Rössle, R.: Die geweblichen Äusserungen der Allergie, Wien. klin. Wchnschr. 45:609-613 (May 13); 648-651 (May 20) 1932.

25. Gruber, G. B.: Zur Frage der Periarteriitis nodose, mit besonderer Berücksichtigung der Gallenblasen- und Nieren-Beteiligung, Virchows Arch. f. path. Anat. 258:441-501, 1925.

26. Delaunay, Albert, Lebrun, Jacqueline, and Delaunay, Marcelle: Le rôle des troubles vaso-moteurs dans la genèse des lésions allergiques, Compt. rend. Acad. d. sc. 226:1110-1111 (Mar. 31) 1948.

27. Goddard, J. W.: Granuloma, Characteristic "Qualitative" Changes in Focal Anaphylactic Inflammation, Am. J. Path. 23:943-960 (Nov.) 1947.

28. Krogh, August: The Anatomy and Physiology of Capillaries, New Haven, Yale University Press, 1922, 276 pp.

29. Selye, Hans, and Pentz, E. Irene: Pathogenetical Correlations between Periarteritis Nodosa, Renal Hypertension and Rheumatic Lesions, Canad. M. A. J. 49:264-272 (Oct.) 1943.

30. Marcus, Henry: Studie über die symmetrische Gangrän. II. Experimentelle Untersuchungen über die Bedingungen der Entstehung der Gangrän, Acta med. Scandinav. 54:413-455, 1921.

31. Gerlach, Werner: Studien über hyperergische Entzündung, Virchows Arch. f. path. Anat. 247:294-361, 1923.

32. Selfridge, Grant: Arterial Spasm and Fat Metabolism: Their Relation to Certain Diseases and to Certain Members of the Vitamin B Complex, California and West. Med. 62:163 (Apr.) 1945.

33. Evans, E. R.: Hypersensitivity, the Common Denominator of the Collagenous Diseases, California Med. 68:335-338 (May) 1948.

34. Harkavy, Joseph: Vascular Allergy, J. Allergy 14:507-537 (Nov.) 1943.

35. Kline, B. S.: Tissue Changes in Allergy, J. Allergy 19:19-34 (Jan.) 1948.

36. Petersen, W. F., and Milliken, Margaret E.: The Patient and the Weather; Autonomic Dysintegration, Vol. 2, Ann Arbor, Edwards Brothers, Inc., 1934, 530 pp.

37. Cannon, W. B.: The Wisdom of the Body, New York, W. W. Norton & Company, Inc. 1932, 312 pp.

38. Cannon, W. B., and Rosenblueth, Arturo: Autonomic Neuro-effector Systems, New York, The Macmillan Company, 1937, 229 pp.

39. Müller, Otfried: Die feiunsten Blutgefässe des Menschen in gesunden und kranken Tagem, Vol. 2, Stuttgart, Ferdinand Enke, 1939, 928 pp.

40. Nedzel, A. J.: Vascular Spasm: Experimental Studies. Vol. 3, no. 3-4, Urbana, Illinois, University of Illinois Press, 1943, 151 pp.

41. Kahn, R. L.: Tissue Immunity, Springfield, Illinois, Charles C. Thomas, 1936, 707 pp.

42. Duke, W. W.: Physical Allergy; Preliminary Report, J. A. M. A. 84:736-740 (Mar. 7) 1925.

43. Opie, E. L.: Pathogenesis of the Specific Inflammatory Reaction of Immunized Animals (Arthus Phenomenon); the Relation of Local "Sensitization" to Immunity, J. Immunol. 9:259-268 (July) 1924.

44. Timonen, S., and Zilliacus, H.: Sludged Blood in Allergy, Acta med. Scandinav. 135:292-297, 1949.

45. Menkin, Valy: Modern Concepts of Inflammation, Science n.s. 105:538-540 (May 23) 1947.

46. Williams, H. L.: A Phylogenetic Concept of Allergy, Proc. Staff Meet., Mayo Clin. 24:516-523 (Sept. 28) 1949.

47. Mayer, R. L., and Kull, F. C.: Influence of Pyribenzamine and Antistine upon the Action of Hyaluronidase, Proc. Soc. Exper. Biol. and Med. 66:392-398 (Nov.) 1947.

48. Robinson, J. J.: Notes on Some Experimental Streptococcus Injury in the Rabbit and Guinea Pig, Arch. Pediat. 61:564-583 (Nov.) 1944.

49. Hawn, C. V. Z., and Janeway, C. A.: Histological and Serological Sequences in Experimental Hypersensitivity, J. Exper. Med. 85:571-589 (June 1) 1947.

50. Klinge, Fritz: Der Rheumatismus; pathologisch-anatomische und experimentellpathologische Tatsachen und ihre Auswertung für das ärztliche Rheumaproblem, Ergebn. d. allg. Path. u. path. Anat. 27:1-351, 1933.

51. Criep, L. H., and Mayer, L. D.: Experimental Collagen Disease from Hypersensitization to Streptococcus Toxin, J. Allergy 20:243-254 (July) 1949.

52. Teilum, Gunnar: Pathogenetic Studies on Lupus Erythematosus Disseminatus and Related Diseases, Acta med. Scandinav. 123:126-142, 1946.

53. Kendall, E. C.: The Adrenal Cortex, Arch. Path. 32:474-501 (Sept.) 1941.

54. Kendall, E. C.: The Influence of the Adrenal Cortex on the Metabolism of Water and Electrolytes. In Harris, R. S. and Thimann, K. V.: Vitamins and Hormones; Advances in Research and Applications, New York, Academic Press Inc. Publishers, 1948, vol. 6, pp. 277-327.

55. Carryer, H. M.: Studies on the Release of Histamine During Hemolytic Reactions in Blood and the Effects of Adrenal Cortical Compound E and Antihistamine Agents in These Reactions. Thesis, University of Minnesota Graduate School, 1948, 80 pp.

56. Sabin, Florence R.: Cellular Reactions to a Dye-protein with a Concept of the Mechanism of Antibody Formation, J. Exper. Med. 70:67-81 (July 1) 1939.

57. Chase, Jeanne H., White, Abraham, and Dougherty, T. F.: The Enhancement of Circulating Antibody Concentration by Adrenal Cortical Hormones, J. Immunol. 52:101-112 (Feb.) 1946.

58. Ehrich, W. E.: The Role of the Lymphocyte in the Circulation of the Lymph, Ann. New York Acad. Sc. 46:823-847 (Sept. 16) 1946.

59. White, Abraham, and Dougherty, T. F.: The Role of Lymphocytes in Normal and Immune Globulin Production, and the Mode of Release of Globulin from Lymphocytes, Ann. New York Acad. Sc. 46:859-880 (Sept. 16) 1946.

60. White, A.: Relation of Adrenals to Immunity, Bull. New York Acad. Med. 24:26-31 (Jan.) 1948.

61. McMaster, P. D., and Hudack, S. S.: The Formation of Agglutinins within Lymph Nodes, J. Exper. Med. 61:783-805 (June 1) 1935.

62. Ehrich, W. E., and Harris, T. N.: The Formation of Antibodies in the Popliteal Lymph Node in Rabbits, J. Exper. Med. 76:335-347 (Oct. 1) 1942.

63. Heilman, F. R., and Kendall, E. C.: The Influence of 11-dehydro-17-hydroxycorticosterone (Compound E) on the Growth of a Malignant Tumor in the Mouse, Endocrinology 34:416-420 (June) 1944.

64. Valentine, W. N., Craddock, C. G., Jr., and Lawrence, J. S.: Relation of Adrenal Cortical Hormone to Lymphoid Tissue and Lymphocytes, Blood 3:729-754 (July) 1948.

65. Selye, Hans: The General Adaptation Syndrome and the Diseases of Adaptation, J. Clin. Endocrinol. 6:117-230 (Feb.) 1946.

66. Brunsting, L. A.: The Effect of Cortisone on Disseminated Lupus Erythematosus. Unpublished data.

67. Shick, R. M., Baggenstoss, A. H., and Polley, H. F.: The Effects of Cortisone and ACTH on Periarteritis Nodosa and Cranial Arteritis: Preliminary Report, Proc. Staff Meet., Mayo Clin. 25:135-143 (Mar. 15) 1950.

68. Carryer, H. M., Koelsche, G. A., Prickman, L. E., Maytum, C. K., Lake, C. F., and Williams, H. L.: The Effect of Cortisone upon Bronchial Asthma and Hay Fever Occurring in Ragweed Pollen Sensitive Subjects. Unpublished data.

XLIX

SCREWWORM (COCHLIOMYIA AMERICANA) INFESTATION IN MAN

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Rarely has a paper been presented before this distinguished senior society dealing with a subject pertaining to tropical or semitropical medicine. Although this is a national society, I do not find it amiss to discuss a disease which at times may hold profound interest for the otolaryngologists residing in the Deep South. It so happens that cases of human infestation with screwworms occurring as far north as Missouri and Illinois have been reported. Also, my subject emphasizes the fact that with the speeding up of rapid air transportation from tropical countries the American physician at times may be confronted with medical problems with which he is unfamiliar, not the least of which may be this repelling parasitic disease.

In 1933, as a result of the dust storms and the forming of dust bowls in some of the Southwestern states, large areas of verdant pasture and grazing lands were destroyed. The United States Department of Agriculture, taking cognizance of this destruction and acting to prevent the starvation of large herds of cattle in these areas, sent thousands of half-starved and emaciated animals to the rich grazing pasturelands of Florida and Southern Georgia. Many of these cattle were infested with the screwworm. Within two years the infestation of this parasite was widely established in this virgin territory, and a high mortality was being reported by the livestock owners. The Bureau of Entomology and Plant Quarantine reports that the loss from this source in the Southwestern states may amount to $10,000,000 annually.

Not until 1937 were cases reported in Florida of the infestation of man with this parasite.[1] My introduction to this cruel affliction was a tragic one, for one of my first cases terminated fatally as the result of meningitis four days after the patient was admitted to the hospital. In another case, the patient narrowly escaped death owing

to massive and repeated nasal hemorrhages. In still another case, there developed a saddle back nose, almost complete loss of the soft palate resulting from cicatricial atresia of the nasopharynx, and a fistula in the maxillary antrum extending through the soft tissues of the cheek. In all of these cases, when the patient was first seen the disease was in the late stages, which accounted for the serious sequelae. I am convinced that the mortality would be relatively as high in man as it is in livestock if the disease were allowed to remain untreated.

A comparatively high mortality as a result of this disease has been reported in the literature. In 1934, Aubertin and Buxton[2] reported 179 cases with 15 deaths. In the series of Yount and Sudler,[3] 23 cases, or 22 per cent, terminated fatally. Harris[4] in 1929 reported 2 deaths in 5 cases.

Time will not permit me to give a detailed report of the 8 cases it has been my fortune, or misfortune, to treat. I have, however, observed most of the complications which are mentioned in the body of this paper.

COCHLIOMYIA AMERICANA

In 1933 Cushing and Patton,[5] in a study of blow flies, concluded that Cochliomyia americana is the true myiasis-producing screwworm fly of the Americas. This fly is a shiny bluish green blow fly distinguished from the ordinary bluebottle fly by three longitudinal black stripes symmetrically arranged on the thorax or scutum. The head is short, red or yellow in color, and closely attached to the body (Fig. 1). Stiff black hairs cover the body, which is metallic in appearance. This specie of fly is small, varying in length from 10 to 13 mm. The female lays at one time from 10 to 400 oblong yellowish eggs (Fig. 2), depositing them on the edges of wounds or blood spots and in body cavities cemented together in shingle-like masses rather than in the haphazard fashion of other blow flies. In from six to twenty-one hours the eggs hatch. The tiny larvae (Fig. 3) known as screwworms, are described as whitish footless grubs that are rather slender and quite active; they feed in clusters and burrow into the live flesh until soon a pocket or cavity is formed. When these worms reach maturity in from three and one-half to ten days, they attain a length of from 13 to 19 mm. and have twelve segments with a belt of minute spines or bristles around each one. They somewhat resemble a screw as the body tapers from

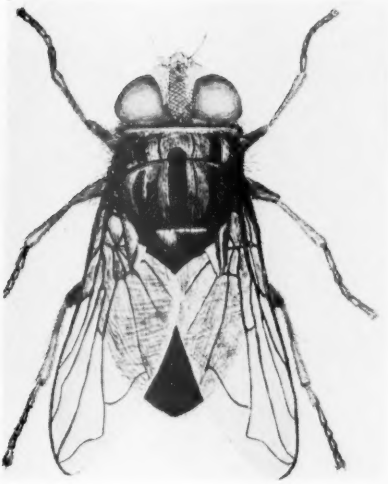

Fig. 1.—Adult screwworm fly (Cochliomyia americana). From Plate 1, Tech. Bul. 500, U. S. Dept. of Agriculture, January 1936.

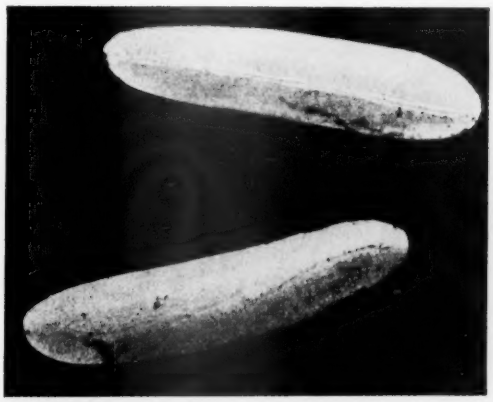

Fig. 2.—Eggs of the screwworm fly.

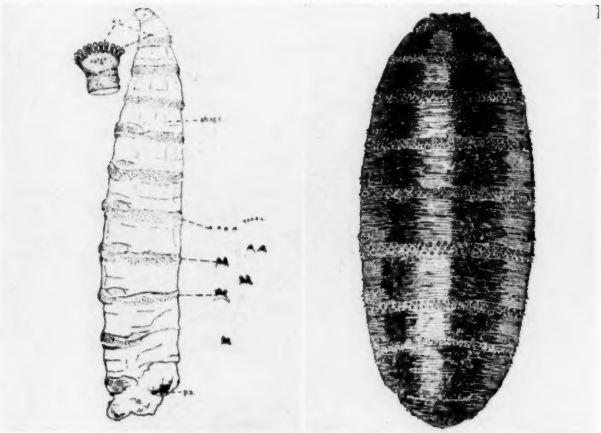

Fig. 3.—Third stage larva of the screwworm. Note the anterior spiracles with eleven finger-like processes and the belts of small bifid or trifid spines (some shown enlarged). From Figure 236, Insects, Ticks, Mites and Venomous Animals of Medical and Veterinary Importance by Walter Scott Patton and Alween M. Evans, Croydon, H. R. Grubb, Ltd., 1929.

Fig. 4.—Pupa of Cochliomyia americana. From Figure 12, page 19, Tech. Bul. 500, U. S. Dept. of Agriculture, January 1936.

the large posterior end to almost a point at the head. Embedded in the living flesh, the head is provided with two hornlike mouth hooks capable of tearing living tissues and causing them to bleed. The posterior end remains uppermost for on it are situated the spiracles or breathing orifices.

On reaching maturity the screwworms drop from the host to the ground, where they burrow into the soil, burying themselves while the skin hardens as they pass through the pupal stage to develop into the adult fly. The pupae are brown, elongated oval in shape and about 10 mm. in length (Fig. 4). Emerging in from seven to fourteen days, the adult flies are ready to mate and to lay eggs after an additional period of from five to ten days. Thus the average life cycle is approximately twenty-four days, and in consequence a number of generations may be bred in a single season.

SITE AND PREDISPOSING FACTORS

The common site of infestation in man is the nose, but infestation of the nasal sinuses, pharynx, throat, mouth, ear, orbit, eyeball and open wounds may also take place. In rhinal myiasis the larvae occur in the nose as facultative parasites, then invade the surrounding tissues and the paranasal sinuses, even penetrating the bone in some instances. Occasionally they attack the drum membrane, cartilage and bones of the ear.

The chief predisposing factor in rhinal myiasis is a pre-existing pathologic condition of the nose. A cut, scratch, or abrasion of the skin is necessary to attract the female screwworm fly. It rarely attacks normal tissue, preferring always an open wound or bleeding surface in which to deposit its eggs. Chronic rhinitis or uncleanliness attracts the female fly, as does also any exposed wound or ulcer. In reviewing the cases described in the literature, one notes the repeated mention of the presence of atrophic rhinitis, ozena and syphilitic disease as contributory factors; also occasionally epistaxis, epithelioma and leprosy are named. The presence of the disease itself invites reinfection. The offensive odor associated with ozena and also the smell of blood appear to attract the fly.

Sleeping in the open invites infestation for the insect may deposit its eggs undisturbed. In numerous cases reported in the literature the patient had slept out of doors. The disease occurs more frequently in farmers and other workers in rural districts than in urban dwellers; it occurs with greater frequency in the poor than in persons in better circumstances, owing doubtless in both instances to their mode of living. Since climate and the season of the year affect the habits of the fly that causes the disease, they also affect its prevalence. The seasonal incidence varies with the latitude, and a larger number of cases naturally occur in the warmer climates where the fly has a longer period of activity and is more numerous.

Within twenty-four hours after the eggs are deposited in a favorable environment they hatch out, and the young parasites penetrate farther up into the nasal cavity, feeding on the discharge caused by antecedent conditions of disease or by the irritation incident to their presence. Their movements, however, may not be perceived by the patient. When opportunity offers, more and more flies are attracted to the infested wound, and the larvae continue to develop from the new batches of eggs. The worms readily penetrate and enlarge the

wound as they rapidly destroy the tissues, until finally vital organs are exposed or sufficient poison is absorbed to prove fatal. Death may then ensue from continual reinfestation or from complications resulting from the presence of these parasites.

SYMPTOMS

Subjectively, the symptoms include partial obstruction to breathing on the affected side and a feeling of discomfort accompanied by a strange sensation at the root of the nose that radiates outward along the supraorbital ridge and gradually increases until it becomes an intense pain, usually centering over the frontal sinuses, but sometimes located in the maxillary region, the temporal region or near the ear. Sneezing may occur at the onset, and severe headache may persist. These early subjective symptoms may be noticed in from one to three days after the eggs are deposited in the nose, and a serosanguineous nasal discharge of offensive odor accompanies or soon follows their appearance. As the disease advances, the discharge becomes more profuse, appears in lesser amount from the other nostril and has a more purulent aspect. It is thicker, dirty reddish brown in color and nauseatingly offensive as to odor. The presence of blood in the discharge indicates the activity of live worms in the tissues. As the patient improves after removal or destruction of the larvae, the blood disappears, and the rapidly diminishing discharge becomes mucopurulent or mucoseropurulent.

With the onset of this discharge, the patient may clear his throat repeatedly, expectorate frequently and cough up a purulent material that may contain living or dead larvae, especially after treatment is instituted. The nose becomes greatly irritated, and the temperature is elevated, ranging from 101 to 104 F. Neuralgia and headache are usually pronounced, particularly if the parasites invade the sinuses. Swelling of the nose, eyelids and cheeks occurs on both sides, but arises first and is greater on the affected side. As the disease progresses, the surface over the swelling becomes discolored and has the appearance of the skin in erysipelas. Destruction of the tissue may even extend to the surface of the face and, by breaking down the skin, cause a foul open sore. In some instances it has been possible to discern the movement of the larvae under the intact skin (Fig. 5). At this stage subjective sensation of activity within the nasal chambers has developed, and the swarming larvae may be observed actively in motion. Their number varies from a few to several

hundred. These parasites may destroy the nasal conchae, perforate the septum and the palate, and make their way into the paranasal sinuses, the orbit, the eyeball, and even the intracranial space and the brain.

Concomitant ocular symptoms may also be present. They include swelling, redness, lacrimation, photophobia, blepharospasm, irritation of the bulbar and palpebral conjunctivas, and the gathering of mucopurulent material at the inner canthi. In one of my cases of nasal myiasis the patient's right eye was swollen shut, and a worm was found in the lacrimal duct (Fig. 5).

Fig. 5.—In this patient the screwworms were swarming throughout the nasal cavity, coming from the antrum through the tooth sockets into the oral cavity, and perforating the soft palate. There was invasion of one tonsilar fossa with pronounced swelling of the superficial tissue. The right eye was swollen shut, and a worm was found in the lacrimal duct. Over the frontal area movement of the larvae was discernible.

The patient suffering from this disease is as a rule extremely nervous and restless at first, and irritated at the inability to clear the nose. As the condition becomes more and more toxic, the apathy that develops is superceded by stupor or even coma. Delirium or a semidelirious state occurs in some cases. These mental symptoms disappear quickly with improvement of the patient.

Pallor and loss of weight have been reported as symptoms of rhinal myiasis. Epistaxis is not only a predisposing cause, but it may occur in the active stage also or as a delayed symptom. It may cause fatal termination.

DIAGNOSIS

Usually it is possible to obtain a history of a fly in the nose two or three days before the onset of symptoms. This information

coupled with the presence of obstruction, intense irritation and an offensive sanguinopurulent discharge from the nose should make the diagnosis of rhinal myasis practically certain, especially in the warmer climates. Observation of the larvae is definitely diagnostic. They may be seen more easily in the initial stage of the disease after the swollen mucous membrane has been shrunk with adrenalin. Later they are readily visible high in the nasal cavity, and as the condition progresses, they may be observed in the nasal fossae, around the artificial openings they have made and in expectoration from the nasopharynx. Once observed, a wound infested by screwworms is easily recognized by the characteristic watery discharge of bloody exudate and the offensive nauseating odor. Also, inspection reveals the worms, standing on end, in the pockets or far recesses with head or small end embedded in the flesh.

Differential diagnosis is not difficult. The presence of a foreign body within the nose might at first be suspected, as might also local inflammatory swelling from some other cause. Even early in the disease, however, careful inspection will disclose the larvae in the nasal cavity; if not, treatment with chloroform will probably bring them to view. The parasites are plainly visible when the disease is fully developed; otherwise, the condition might appear to simulate acute paranasal sinusitis, anthrax, erysipelas, or cellulitis of the face.

COMPLICATIONS AND PROGNOSIS

Since the destructive process may progress into the adjacent structures as the larvae migrate into the surrounding tissues or sinuses, complications are not infrequent. Paranasal sinusitis is doubtless the most frequent involvement with the frontal sinuses commonly affected. Migration of the larvae into the orbit or into the cranial cavity sometimes occurs. Once the brain is reached, secondary bacterial invasion causes fatal termination. Hemorrhage may also be so extensive as to prove fatal. Otitis media with suppuration has been reported, as has invasion of the throat. Inspiration pneumonia is occasionally encountered. Gastric and intestinal myiasis occur but seldom as complications or sequelae, and septic meningitis and general sepsis also occur rarely.

In severe cases that do not terminate fatally, the patient may suffer varying degrees of deformity owing to the destruction of tissue. In some instances it may even be necessary to resort to plastic surgery as a remedial measure. Saddle nose, perforated septum, per-

forated palate or destruction of portions of the nose or cheek may occur. The conchae and nasal septum are usually attacked. In cases in which ozena is a predisposing factor, destruction by the larvae of portions of the mucous membrane lining the nasal cavities aggravates this disease. If the larvae make their way to the eyeball, loss of the eye may result. The outlook, however, is favorable when only a few larvae are present and treatment is promptly instituted. In the absence of complications, reinfections or sequelae, the period of disability is relatively short for the larvae mature in a few days.

TREATMENT

In the treatment of rhinal myiasis removal of the larvae is accomplished mechanically and by the use of drugs. It is important to cleanse the wound thoroughly so that the spiracles or breathing orifices on the posterior end of the embedded larvae may be exposed to the fumes of the drug used and so that the drug itself is permitted to reach the worms submerged in the pus. Forced to breathe the fumes through the exposed spiracles, the worms are stupified in a few seconds and killed after several minutes. Chloroform applied locally and as a vapor is the drug of election.

Symptomatic treatment includes measures for the relief of pain, nervousness, insomnia and anxiety. It is important to enforce general hygienic rules and to provide stimulative and supportive therapy when required. Because of the repulsive nature of the disease, anorexia is commonly experienced by patients whose minds are clear, and liquid or semiliquid foods are often prescribed.

The treatment and, in so far as possible, the cure of intranasal pathologic conditions are highly important as preventive measures. The patient suffering from ozena, for example, should be made aware of the danger of myiasis and should be cautioned against sleeping in the open unprotected by screens. He should be informed regarding the immediate need for medical attention in case a fly enters the nose.

Basically, sanitary provision for the prompt and suitable disposal of refuse and carcasses, thus preventing to a considerable degree the breeding of the fly whose larvae cause myiasis, is a constructive preventive measure. This pest has a more devastating effect on both domestic and wild animals than any other insect; uncontrolled, its larvae are capable of wiping out entire herds of cattle, hogs, sheep

and goats. They attack any warm-blooded animal for they are true parasites that take up their abode only in the living flesh of such an animal. Infestations have been observed in all kinds of wild and domestic animals and in poultry, as well as in man. Thus the danger of myiasis is not to be overlooked, particularly in the presence of the predisposing factors described.

SUMMARY

Infestation of the nasal cavity by the screwworm (C. americana) is described. The symptoms, differential diagnosis, complications, prognosis and treatment are discussed. Mention is also made of the relatively high mortality and of the predisposing factors associated with this disease in man.

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REFERENCES

1. Taylor, H. M.: Screwworm Infestation of Man, Bulletin of the Riverside Hospital (Jacksonville, Fla.) 2:33 (July) 1938.

2. Aubertin, D., and Buxton, P. A.: Cochliomyia and Myiasis in Tropical America, Ann. Trop. Med. 28:245, 1934.

3. Yount, C. E. and Sudler, M. T.: Human Myiasis from the Screw-Worm Fly, J. A. M. A. 49:1912, 1907.

4. Harris, S. T.: Human Myiasis, U. S. Vet. Bur. Med. Bull. 5:412, 1929.

5. Cushing, E. C., and Patton, W. S.: Studies on the Higher Diptera of Medical and Veterinary Importance, Ann. Trop. Med. 27:539, 1933.

L

SMOKER'S LARYNX

A CLINICAL PATHOLOGICAL ENTITY

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The purpose of this paper is to make known a lesion of the vocal cord which is caused directly by the irritation of smoking. At first the lesion is purely that of an inflammatory reaction. The earliest change to be noted is a localized edema of part of a vocal cord. Later this edematous area gives way to a well established neoplasm, which histologically, is an edematous fibroma. This is the direct result of irritation from smoking.

The first response to a local irritant is a flow of serum into the irritated area. If the irritant is removed, the edema disappears. If the irritation is continued a chronic edema results. It is a well established fact that chronic edema anywhere in the body favors fibrosis. The fibrous tissue which is laid down changes the consistency and size of the mass. New blood vessels are formed, but throughout the life history of the lesion, the outstanding picture is that of edematous fibroma. Even after many years when there is considerable vascularity on section, the color has changed but slightly to a light pink from the usual pearly grey. As a result of the increased fibrous tissue the tumor assumes increased firmness. The firmness of the tumor now causes a different kind of irritation of the opposite vocal cord; this, in addition to the continuously added insults from the irritating smoke. If the individual continues to smoke he soon will have masses on both cords; in some cases they are multiple, depending upon the intensity and duration of exposure to the irritant.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Read before the meeting of the American Laryngological Association, San Francisco, California, May 24, 1950.

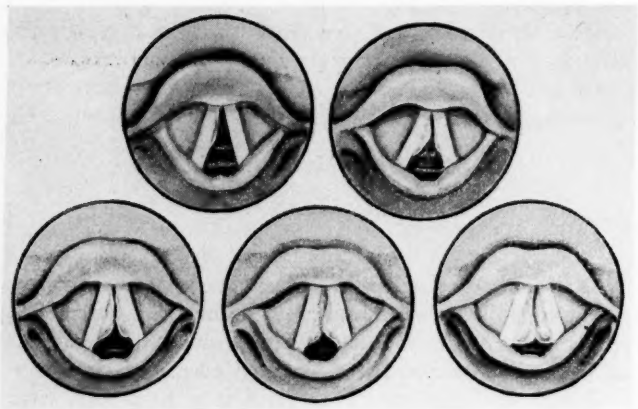

Fig. 1.—Edematous Fibromata—development from earliest to latest stages.

Histologically the sections contain nodules composed of fibrous connective tissue in an edematous stroma. In some areas there is a heavy infiltration of inflammatory cells, chiefly plasma cells, lymphocytes and polymorphonuclears. In cases of long standing one sees hyalinized collagen in the stroma. The epithelium is usually stratified squamous; it may show ulceration, atrophy, acanthosis or any of the surface changes to which laryngeal neoplasms are prone.

Edematous fibroma gives the appearance of a polyp or polypoid tissue, such as is seen in the noses of allergic individuals. Although nasal polypi depend upon localized edema for their formation the histological picture is quite different. In the nasal polyp there is a definite, thickened basement membrane and the stroma shows a heavy infiltration with eosinophiles. Such is not the case with the edematous fibroma.

The size and shape of edematous fibroma varies. The variation is due to the duration of the lesion and the intensity of exposure to the irritating factors in smoking. As a rule the earliest lesions are unilateral and relatively small, rarely occupying more than one half of the anterior two thirds of the vocal cord. The lesions are always sessile in their attachment. The uninvolved side sooner or later will have a similar edematous fibroma in its anterior portion, if smoking is continued as before.

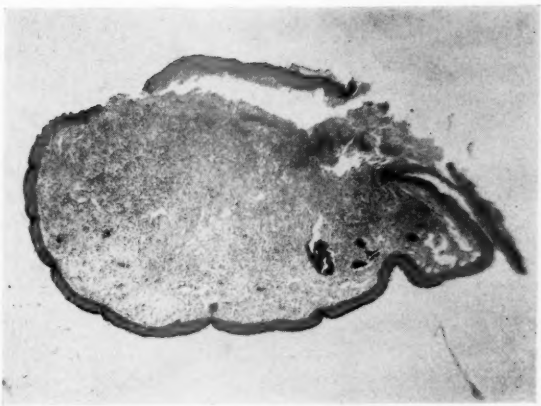

Fig. 2.—Photomicrograph—low power, of early edematous fibroma. The mucosa appears to be normal laryngeal. The stroma is intensely edematous; scattered throughout the stroma are inflammatory cells, plasma cells, polymorphonuclear leukocytes, and some lymphocytes.

After an edematous fibroma has lasted for some time there is a gradual increase in size, both at the site of attachment and in the body of the tumor itself. Because of the consistency of these growths the larger ones are inclined to drop between the vocal cords, especially during inspiration. In as much as edematous fibroma is essentially an anteriorly placed tumor, obstruction to the airway is most unusual.

When small, edematous fibroma is seen as a flat ovoid mass continuous from the part of the vocal cord from which it springs. When larger, however, it is still ovoid in shape but looks more like a polyp.

On four occasions I have had the opportunity to observe a localized edematous area of one vocal cord in individuals who have been smoking excessively. In each case the edema disappeared within twenty-four hours, when the source of irritation was withdrawn.

Laryngologists are well acquainted with the submucous space of the vocal cord, Reinke's space; they are well aware of its capacity to accumulate fluid as a result of reaction to any type of irritation.

This paper is based upon one hundred and forty-three patients who presented themselves with one or more swellings of the vocal

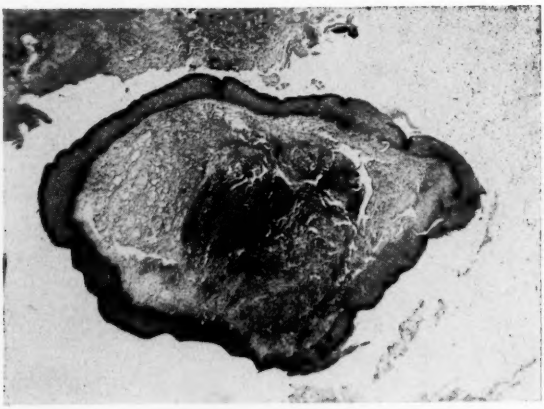

Fig. 3.—Protomicrograph—low power, of edematous fibroma showing long lasting and late stage of this condition. The mucosa is extremely thickened, having flattened outer layers; there is a tremendous increase of fibrous tissue in addition to considerable hyalinized collagen. Inflammatory cells are numerous.

cords as a result of excessive smoking. Except for three who were cigar smokers, they smoked from 20 to 120 cigarettes daily. Most smoked 40 or more. Four were seen with an early localized edematous lesion of one of the cords. In each of these the edematous area disappeared within twenty-four hours as a result of abstaining from smoking.

Of the one hundred and forty-three, fourteen were females, the remainder were males. Their ages ranged from sixteen to sixty-nine years of age.

The clinical manifestations of edematous fibroma of the vocal cord are not unusual. They are the same as for any group of vocal cord tumors. Hoarseness of varying degree, and vocal fatigue are the usual symptoms. The longer the duration, the firmer the growth and the more hoarse the individual. As in other types of neoplasms the more anterior, the greater the degree of hoarseness. Obstruction of the larynx as a result of edematous fibroma is extremely rare. One of the patients in this series required a tracheotomy. He was a forty-nine year old truck driver who smoked between five and six packages

of cigarettes a day. He drove a truck across country, mostly at night. He explained that he had nothing much else to do than smoke on these long trips. He had had a raspy voice for several years and had been markedly hoarse for more than two years. One night he was unable to breathe. He struggled for air and was threatened with asphyxia. Had it not been for the artificial respiration administered to him three times during the night by his son he would have expired. When seen by me the next morning he was sent to the hospital for immediate tracheotomy, which was performed with considerable difficulty, because of the marked obstruction to the airway and the short muscular neck. A week after tracheotomy a large number of edematous fibromata were removed by indirect laryngoscopy. The patient was sent home with a clean larynx and a good voice.

Of interest is the fact that a great majority of the men who are seen with edematous fibroma have short muscular necks. It may be that the short neck favors vascular changes in the larynx.

The diagnosis is made on a basis of the appearance of the lesion and a history of excessive smoking. This condtion must be differentiated from a localized tuberculous edematous infiltration of the vocal cord. Such a lesion can be identical in appearance with that under consideration; it is therefore important that a careful history be taken to eliminate the possible presence of tuberculosis. X-ray studies of the chest should always be made. It is also necessary to have every mass of this type which is removed from the larynx studied histologically.

The treatment of smoker's larynx or edematous fibroma of the vocal cord is removal of the mass. Only in the very early stage when the lesion is purely edematous can conservative or non-surgical treatment be used. As has been previously noted, abstinence from smoking for twenty-four hours will cause a return of the voice and vocal cord to normal. The practice of placing patients with these vocal cord lesions on vocal rest for two or more weeks is based on a faulty conception of the disease. If the lesion does not disappear within twenty-four hours it can be safely considered permanent.

Removal is accomplished by means of indirect laryngoscopy. This procedure is simple, free from physical or psychic injury and does not require special assistants. The use of an operating room is optional. The neoplasms are removed with the patient sitting in a

chair. His larynx is anesthetized and the tumors are removed with a curved instrument to the end of which is attached a Frankel tip, a double cup type of forceps. Any benign tumor of the vocal cord can be removed with this type of forceps without injury to the patient. Those who have experienced both indirect and direct laryngoscopy are impressed with the simplicity of the former procedure.

Most edematous fibromata come away from the vocal cords readily. When the growth contains considerable fibrous tissue, part of the matrix is liable to be left behind with the first attempt at removal. A second or third application of the forceps is then necessary to successfully complete the operation.

SUMMARY

A localized lesion of the vocal cord which is due to excessive smoking has been described. Its genesis and development can be observed in many cases. Histologically, this lesion is one of localized edema which becomes chronic. As a result of the chronic edema fibrous connective tissue forms in the stroma and an edematous fibroma results.

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REFERENCES

1. The Tobacco Situation; Bureau of Agricultural Economics, United States Department of Agriculture, October, p. 6, 1949.

2. Annual Report on Tobacco Statistics; United States Department of Agriculture, Washington, D. C., December, p. 77, Table 24, 1942.

3. Haag, H. B.: Studies On The Physiologic Action of Diethylene Glycol, J. Lab. and Clin. Med. 22:341, 1937.

4. Finnegan, J. K., Fordham, Doris, Larson, P. S., and Haag, H. B.: A quantitative Method for the Measurement of Cigarette Smoke Irritation, J. Pharmacol. and Exper. Therap. 89:115, 1947.

5. Larson, P. S., Haag, H. B., and Finnegan, J. K.: Studies On Cigarette Smoke Irritation; I. Determination of Edema-Producing Properties of Certain Types of Cigarette Tobaccos; Proc. Soc. Exper. Biol. and Med. 64:476, 1947.

6. Finnegan, J. K., Larson, P. S., and Haag, H. B.: Studies on Cigarette Smoke Irritation, II. The Role of Nicotine, Proc. Soc. Exper. Biol. & Med. 65:200, 1947.

LI

PAPILLOMA OF THE LARYNX: A REVIEW OF 109 CASES WITH A PRELIMINARY REPORT OF AUREOMYCIN THERAPY

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Papilloma of the larynx is one of the most common benign laryngeal neoplasms. It is a common laryngeal tumor of childhood, and is only slightly less frequent in adults. Yet in spite of its frequency, chronicity and multiple recurrences, a search of the literature shows little agreement among various authorities regarding important aspects of the disease. In 1922 Crowe and Breitstein[1] published a review of the literature. New and Erich[2] in their comprehensive article on Benign Tumors of the Larynx reviewed the subject of laryngeal papilloma in 1938, while in 1944 Ferguson and Scott[3] added an additional series of cases and again reviewed the literature. This paper consists of a presentation of 109 consecutive cases of papilloma of the larynx with a discussion of some of the controversial points of etiology, pathological diagnosis, and treatment as determined by a review of these cases.

PRESENTATION OF CASES

The basis of this report consists of 109 cases of papilloma of the larynx seen in the past 15 years (1935-1950) at The Children's Memorial Hospital, Research and Educational Hospitals of the University of Illinois College of Medicine, and St. Luke's Hospital, Chicago.

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ETIOLOGY

The specific etiology of papilloma of the larynx is not known. While there has been no extensive work on this subject, Ullman[4] was apparently able to transplant the growth by means of a filterable agent, possibly a virus, to both the skin and mucous membrane surfaces. Shope and Hurst[5] working with rabbit papilloma have discovered a papilloma virus which they were able to transmit to laboratory animals.

Chronic laryngeal irritation has also been thought to be a possible etiological factor, but histopathologically the lesion is definitely neoplastic and there is nothing to suggest a chronic inflammatory process. However, plantar warts, of a similar histologic character, are considered to be caused by chronic irritation and inflammation, and this possibility must be taken into consideration.

The disappearance of many childhood papillomas of the larynx at the time of puberty has long been a recognized clinical fact. It has been suggested that this indicates that a hormonal factor may be present which causes an epithelial change resulting in disappearance of the lesions. Lewis[6] noted that local applications of esterogenic hormones will produce definite changes in the vaginal mucosa of young girls, changing it from the simple squamous epithelium to the stratified squamous adult type epithelium. Reichert[7] confirmed Lewis' work and demonstrated the value of establishing such changes in the treatment of gonococcal vaginitis in infants.

In favor of this concept of etiology, Broyles[8] states that in five cases of papilloma of the larynx treated topically with estrogenic hormones, the lesions disappeared within six months. No late follow-up reports on these cases have appeared in the literature, and similar observations by other authors have not been made. This work is of extreme interest, however, and deserves further study. While other aspects of this form of therapy are discussed below in the section on treatment, one patient of our series is reported here because of a disappearance of her multiple papillomata during each of a series of three pregnancies, a fact which may indicate the importance of the hormonal factor in the etiology.

Case report: (Case 4) M. N., 25 year old white female was first seen at Research and Educational Hospital in April 1936 because of persistent hoarseness and dyspnea. Multiple papillomata of the lar-

TABLE I.—AGE AT ONSET OF SYMPTOMS.

CHILDREN		ADULTS	
AGE OF ONSET	NO. OF PATIENTS	AGE OF ONSET	NO. OF PATIENTS
0 - 1 year	12	16 - 20 years	2
1 - 2 years	8	21 - 30 "	15
2 - 3 "	5	31 - 40 "	9
3 - 4 "	4	41 - 50 "	14
4 - 5 "	3	Over 51 "	10
5 - 6 "	5	Unknown*	5
6 - 7 "	2		
7 - 8 "	1	Total No. of Adults (over 16 yrs. of Age)	55
8 - 9 "	4		
9 - 10 "	2		
10 - 15 "	8		
Total no. of Children (below 16 yrs. of age)	54		

*Exact age of onset unknown. From history, presumed to be during adult life.

ynx were found, and 63 endoscopic procedures have been performed for forceps removal over a period of 13 years. During the time that this patient has been under observation for papilloma of the larynx she has had three children. She had two children prior to the onset of the papilloma. During the course of each pregnancy the papillomata disappeared; she had a normal voice and no difficulty in breathing. With the onset of menstruation after each pregnancy there was a recurrence of the papilloma. For three years prior to the birth of the last child the patient had no menstrual periods and assumed that she had gone through the menopause. During this time there was no difficulty in breathing and the voice was normal. With the onset of menstruation after the last pregnancy there has been a recurrence of the papilloma.

For the present the etiology of laryngeal papillomata remains in doubt; the most likely possibility appears to be that it is due to a filterable virus, with a hormonal factor influencing the course of the disease.

Age, Sex and Race Incidence: Age of onset of symptoms as clearly as could be determined from the histories of the 109 cases herein presented ranges from the newborn period to advanced adult life, the oldest patient in this series being 72 years of age. In 54 patients (49%) the onset occurred below 16 years of age (Table I). Further examination of Table I shows that 32 of the 109 patients (29%) developed symptoms before reaching five years of age. The sex incidence of the entire group shows an almost equal incidence of males (59) and females (50). The sex incidence of the group below 16 years of age was 25 males and 29 females; that in the group above 16 years of age was 34 males and 21 females. One hundred and two of the patients were white and seven were colored; this figure agrees roughly with the general population distribution.

PATHOLOGY

Grossly the tumors are glistening, elevated, mulberry-like, nodular masses which vary in color from a whitish pink to red (Fig. 1). They may occur anywhere in the larynx, but chiefly on the true and false cords and in the anterior commissure. Frequently they extend subglottically and occasionally into the trachea and bronchi or upwards on the epiglottis, pharyngeal wall, tonsil and soft palate. They vary in size from small nodules to sessile placques or large nodular masses the size of a cherry. The tumors are usually friable and bleed easily with slight trauma, a quality which makes difficult complete removal by a single operation.

Microscopically the papilloma tissues are sessile or papillary structures composed of a vascular connective tissue core covered by stratified squamous epithelium in many layers. There are usually secondary and even tertiary stalks of vascular fibrous tissue covered by the epithelium. Cells in mitosis are frequent, indicating growth activity, but the cells are well differentiated, mature epithelial cells. The growths have no tendency to invade the stroma or submucosa. No histologic difference is recognized between the papilloma of adults and those in children. There was no histologic difference noted between the papilloma of the adults and children as determined by a study of the papillomatous tissue removed from the patients of this series. However, in spite of the absence of a histologic difference, a distinct clinical difference seems to be present. The papillomata in adults seem to be more friable, leaving a cleaner surface on forceps

TABLE II.—SINGLE OR MULTIPLE PAPILLOMA ACCORDING TO AGE GROUPS.

	SINGLE	MULTIPLE
Children (under 16 years of age)	10	44
Adults (over 16 years of age)	35	20
TOTAL	45	64

removal, whereas in children the tumors appear to be more deep seated and have a tougher consistency.

According to the literature,[3, 9, 10] papillomata are multiple in children, whereas in adults they are more frequently single. Of the 54 patients in our series in whom the onset of the disease occurred below 16 years of age, 44 (81%) were found to have multiple papillomata and in 10 (19%), the papilloma was found to be single. Of the remaining 55 patients in whom the papilloma had its onset in postpubescent life, the papillomata were multiple in 20 (36%) and single in 35 (64%) (Table II).

Of further significance is the rate of recurrence in the two groups. Examination of Table III shows the number of surgical procedures required by patients of this series according to the age of onset. It will be noted that 33 of the patients required only one operative procedure, 48 required between two and five operations, 15 required six to ten, and the remaining 13 patients required eleven or more operations because of recurrence of the papilloma. Dividing this further, of the 54 children whose papillomata began before 16 years of age, ten required only one operative procedure, 21 between two and five operations, 13 required six to ten, and the remaining ten patients required eleven or more operations because of recurrence. Of the 55 adults whose papillomata began after 16 years of age, 23 required only one operation, 27 between two and five operations, two required six to ten, and the remaining three required eleven or more operations because of the recurrence of the papillomata.

In this entire series, no patient has developed a malignant degeneration of the papilloma. According to New and Erich[2] who record 194 cases of papilloma of the larynx, "Papillomas can undergo ma-

lignant change. This was noted in three of our cases in which there had been no previous therapy which might account for the development of malignancy. In all these cases a squamous cell epithelioma, grade 1, was found in situ in a mass of papillomas. Removal of the papillomas cured the three patients." Jackson and Jackson[9] state, "Of two hundred and five cases of papilloma of the larynx in adults in our clinic we have seen cancer develop at the same site in six cases (3 per cent)." de Sanson and Amarante,[11] on the other hand, report several cases of papilloma of the larynx which underwent malignant transformation, and imply that papilloma is a transitional form towards carcinoma. In this regard, the statistics of Cunning[10] are of interest. He states, "Occasionally these papillomas invade the underlying structure (submucosa) and change characteristics, which eventually results in their assuming a true malignant form. This is especially true of recurrent multiple papillomas." He classifies papillomas as papillomas of childhood, simple papillomas, and hyperkeratotic papillomas. Of the latter, he records 38 or 14% of 276 patients as occurring with malignant change. It would appear that this significant and rather contradictory variation in the statistics of New and Erich, Jackson and Jackson, and the cases of this series, as opposed to those of deSanson and Amarante, and Cunning, must be on the basis of pathologic terminology or classification rather than on an analysis of identical groups of cases.

SYMPTOMS AND FINDINGS

The symptoms of papilloma of the larynx are the result of the alterations of laryngeal function caused by the tumors. They may be divided according to the changes of respiration and phonation. Changes of phonation usually occur first, with a change in the character of the voice followed by progressive hoarseness or huskiness, and finally aphonia. Respiratory changes are those of croupy cough, stridor, dyspnea, cyanosis and ultimately asphyxia. Compensatory phenomena to aid the patient with a slowly obstructing airway may occasionally be observed. A change in personality in children is not infrequent as they learn they are unable to play actively. They become slow-moving, read rather than play, and remain alone as much as possible. They begin to actively use the accessory muscles of respiration, and indrawing of the supra- and infra-sternal notches becomes pronounced; funnel breast developed in two of the children in this series that had had long standing laryngeal obstruction be-

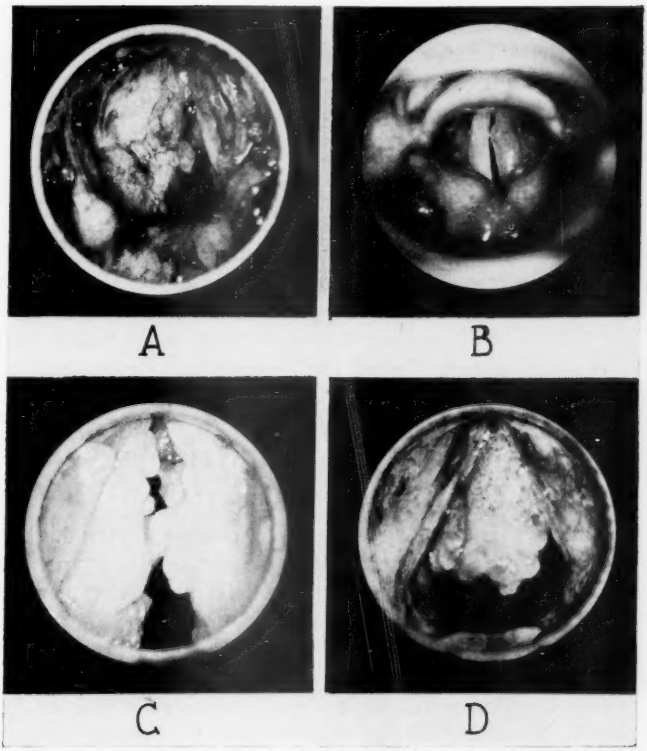

Fig. 1.—Gross appearance of papilloma of the larynx. *A.* Multiple papilloma of the larynx in a child (direct laryngoscopy). *B.* Solitary papilloma of the left vocal cord of a 13 year old girl (mirror laryngoscopy). *C.* Multiple papilloma of the larynx of an adult (direct laryngoscopy). *D.* Large solitary papilloma of the larynx of an adult (direct laryngoscopy).

ginning in early childhood. Occasionally an elevated red blood cell count was noted.

DIAGNOSIS

The diagnosis of papilloma of the larynx depends on the gross appearance of the larynx, either by mirror or direct laryngoscopy, and on the histologic examination of the papillomatous tissue. All benign and malignant lesions causing hoarseness must be considered

in the differential diagnosis. In adults carcinoma, leukoplakia, hyperkeratosis, tuberculosis and syphilis are the principal lesions to be considered. In children the congenital anomalies of the larynx such as webs, cysts and congenital laryngeal stridor are most important, while polyps, vocal nodules, "screamers nodes" and chronic laryngitis are common to both age groups. In limiting the diagnosis of papilloma of the larynx to those lesions which are truly papilloma from the histological standpoint, namely, epithelial tumors in which the cells cover finger-like processes or ridges of stroma, numerous patients with polyps of the larynx are excluded from this series who were originally referred for direct laryngoscopy because of "a papilloma on one of the vocal cords." The loose use of the term "papilloma" in this manner is undoubtedly a confusion with the gross descriptive term "papillary", indicating an elevated nipple-shaped lesion on the edge of the cord. The importance of accuracy in the use of these terms is evident because of the specific clinical significance which is implied by the term "papilloma of the larynx."

TREATMENT

The fact that there is such a wide variation in methods of treatment used in the management of papilloma of the larynx indicates that none has given entirely satisfactory results. Complete reviews of therapeutic procedures used may be found in the excellent papers of Crowe and Breitstein,[1] Foster,[12] Cohen,[13] and Ferguson and Scott.[3] The methods of therapy that have been used to eradicate the papilloma may be divided into the surgical, the medical and the physical measures. These are listed as follows:

I. Surgical
   - A. Forceps removal
   - B. Cautery, electro-coagulation, fulguration and diathermy
   - C. Thyrotomy and laryngo-fissure
   - D. Tracheotomy (to place the larynx "at rest" as well as to establish the airway.)

II. Medical
   - A. Topical medications
     1. Fuming nitric acid, lactic acid, trichloracetic acid
     2. Absolute alcohol, formaldehyde, castor oil
     3. Estrogens—Amniotin
     4. Podophylin

B. Internal Medication

1. Calcined magnesia (magnesium oxide), bismuth compounds and heavy metals, arsenic, potassium iodide
2. Tincture of Thuja
3. Androgens—Testosterone, Testosterone Proprinate, Methyl Testosterone
4. Aureomycin

III. Physical

A. X-ray

B. Radium and Radon—endolaryngeal and extralaryngeal

Various combinations of the above therapeutic procedures and techniques have been employed. Forceps removal frequently has been followed by cauterization, topical applications of medications, or by x-ray or radium therapy. Tracheotomy, when used in combination with other procedures, serves to establish and maintain the airway and, according to early authors on the subject, is of value in limiting the growth of the lesion, by placing the larynx at rest.

In the series of cases herein presented, the majority were treated by means of forceps removal alone. In children this was usually done at frequent intervals without anesthesia, although general anesthesia was employed for extensive removal of papilloma when the growth became profuse and could not be controlled by the other method. In extreme cases in children this has required direct laryngoscopy as many as 136 times in one child over a period of four years. Four of these procedures were done under general anesthesia and the remainder without anesthesia. In a second child, 95 direct laryngoscopies were performed over a period of four years, five under general anesthesia and the remainder without anesthesia. In one adult patient, Case No. 4 previously cited, 63 direct laryngoscopies have been performed; this patient is still under treatment. The first of these three patients was tracheotomized early in the course of treatment and has since been extubated. The other two have not been tracheotomized. In the entire series only 19 of the patients have required tracheotomy—16 of these were under 16 years of age, the remaining three were in the adult group. One of the three adults required tracheotomy because of cicatricial stenosis fol-

lowing X-ray therapy. Papilloma have continued to recur in the larynx of this patient in spite of the fact that sufficient x-ray therapy had been given elsewhere to cause the stenosis which necessitated the tracheotomy.

Cautery has been used only occasionally in patients in this series. However, three patients have been seen with extensive laryngeal webs and stenosis as well as with recurrence of their papilloma who gave histories of previous procedures with cautery elsewhere.

The reports of Broyles[8] stimulated the use of estrogens and androgens in the treatment of laryngeal papillomatosis. The clinical basis for this work, as discussed above, rested on the observation that the papilloma of the larynx of children usually disappeared at puberty. Broyles' report dealt with five cases of papilloma of the larynx in children. The treatment was undertaken with four cases that had received other previous treatment, and one new case. They were treated with weekly applications of the estrognic hormone, Amniotin in oil, 10,000 International units/cc (Squibb). The applications were made through the direct laryngoscope or by spraying the solution directly into the larynx. According to Broyles' report, all five cases were free of papilloma in approximately six months. No secondary sex changes were noted. About 1,000 International units, or 0.1 cc, of Amniotin was applied at each treatment. At present Broyles[14] suggests application of the Amniotin with a laryngeal spray once or twice daily and in addition direct local application at the time of the direct laryngoscopy following forceps removal of the papilloma.

One adult in our series had been treated elsewhere with methyl testosterone orally. He had been under treatment for 18 months with no regression of the papilloma. A single thorough forceps removal has resulted in relief of symptoms and although he is still under observation there has been no recurrence for the past 13 months.

Two patients in this series were treated with Podophyllin (25% in mineral oil). In both cases an exuberant overgrowth of papilloma was observed, with extension into the trachea in one of the children. A similar experience was reported by Barretto[15] following the use of Amniotin.

Greatest controversy in treatment revolves around the use of irradiation therapy. Foster[12] advocates its use but insists it must be

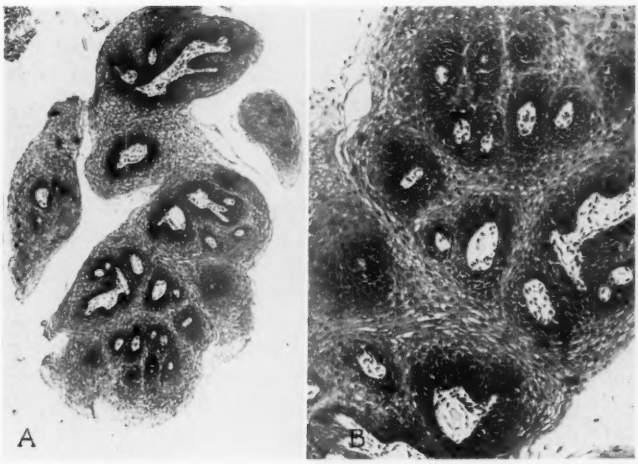

Fig. 2.—Microscopic appearance of papilloma of the larynx. *A.* Low power (X35). *B.* Medium power (X200).

used with low voltage and in mild dosage. Many other reports in the literature suggest favorable results from this type of therapy.[13, 16, 17, 18] The serious complications that have occurred, however, have indicated that this is a dangerous method of treatment. This is especially true when the x-ray therapy has been used in children where destruction of the laryngeal cartilages and arrested development have followed its use, as Salinger[19] and Clerf[20] have indicated in their case reports. The laryngeal destruction, arrested development and chronic laryngeal stenosis that have developed are late results, seen by the laryngologist years after the x-ray therapy and thus are seldom known to the radiologist who administered the treatment and had felt that the papilloma had been cured. More justification for the use of x-ray therapy may be present in the treatment of adults if recurrences are excessive. However, usually less than five surgical procedures suffice to eradicate the lesion in adults, as indicated in our statistics (Table III), and the voice following surgical removal is clear. One adult patient in this series required x-ray therapy because of a failure of forceps removal to control the regrowth. The lesion has apparently been controlled during the past year and a half since receiving irradiation.

The use of aureomycin in the treatment of papilloma of the larynx was suggested[21] because of the specificity of this chemotherapeutic agent towards the virus infections. Seven children ranging in age from one to nine years have received aureomycin with encouraging results. The following are brief summaries of these seven cases (Table IV).

L. K. (Case 92) 3 year old white male, hoarseness two years, dyspnea two and a half months. Original examination on March 25, 1949 showed marked laryngeal obstruction due to exuberant papilloma. Seven forceps removals of papilloma had been made prior to aureomycin therapy. A tracheotomy was necessary on November 11, 1949 because of closure of the larynx by papilloma. A thorough removal of the papilloma under general anesthesia was accomplished on November 21, 1949, and aureomycin therapy instituted; 50 mg. per kg. per day were administered to December 8, 1949 (18 days). Direct laryngoscopy December 12, 1949 revealed the papillomatous tissue had disappeared. On January 6, 1950, a small area of irregularity on the left cord was seen and the general appearance of the larynx was satisfactory and free of papilloma. This patient has moved elsewhere and further immediate follow-up seems unlikely. Nevertheless, the improvement noted was striking and at variance with the appearance following previous forceps removal.

H. T. (Case 104), 4 year old colored male was first seen in November 1949, with a history of hoarseness and difficulty breathing for two months. Examination revealed the larynx to be entirely filled with papillomata and the airway was markedly obstructed. On November 19, 1949, under general anesthesia, the papillomatous material was removed by forceps and the airway markedly improved. Following this operative procedure aureomycin was administered in a dosage of 125 mgm. every six hours, approximately 50 mgm. per kilo., for sixteen days. On December 10, 1949, the day following cessation of therapy, examination of the larynx showed it to be free of papilloma and the cords only slightly red with a faint granular appearance. On May 3, 1950 re-examination revealed a very small papilloma on the left arytenoid; the cords were white and normal in appearance and the airway was normal.

J. S. (Case 61), 9 year old white male, was first seen in September 1946 with a history of hoarseness for two months. Examination revealed a papilloma occupying the entire left cord. Between Sep-

TABLE III.—NUMBER OF OPERATIVE PROCEDURES ACCORDING TO AGE AT TIME OF ONSET OF SYMPTOMS.

Age at Onset	Tumor-free after one operation	2-5 Operative Procedures	6-10 Operative Procedures	11-15 Operative Procedures	16-20 Operative Procedures	Over 20 Operative Procedures
0 - 1	1	5	2	1	1	2
1 - 2		2	5			1
2 - 3		4	1			
3 - 4		1	3			
4 - 5	1	1				1
5 - 6	1	3	1			
6 - 7		1			1	
7 - 8	1					
8 - 9	1	1		1		1
9 - 10	1			1		
10 - 15	4	3	1			
16 - 20		1	1			
21 - 30	5	8	1			1
31 - 40	4	5				
41 - 50	5	8			1	
Over 51	6	3		1		
*Unknown	3	2				

*Exact age of onset unknown. From history, presumed to be during adult life.

tember 30, 1946 and December 30, 1949 ten endoscopic procedures were required for forceps removal of the papilloma. As the disease progressed more structures of the larynx were involved so that on December 30, 1949 the entire larynx was filled and the airway somewhat reduced in size. Following the removal on December 30, 1949, he was placed on aureomycin therapy, a dosage of 500 mgm. four times daily for fourteen days. On March 3, 1950 a tracheotomy was necessary because of recurrence of the papilloma. On April 9, 1950 the larynx was examined and papilloma again removed with forceps. He is receiving another course of aureomycin, 500 mgm. four times daily for 21 days, and will be observed again in one month.

L. B. (Case 106), 5½ year old colored male was first seen in January 1950 with history of hoarseness for three years and dyspnea on exertion. Examination revealed the larynx to be filled with papilloma, especially anteriorly. The papilloma were removed with forceps under general anesthesia and aureomycin therapy instituted. He received 100 mgm. five times daily for a period of 28 days. Five days following cessation of therapy the larynx, on direct examination, was markedly improved and the papilloma had almost entirely disappeared. Two small areas of papilloma the size of a match head were present, one on the midportion and the other at the anterior tip of the left cord. No further therapy was administered. Examination on May 19, 1950 revealed a clear voice, and normal appearance of the vocal cords.

R. DuR. (Case 109), 2 year old white male was first seen in February 1950 with a history of hoarseness since birth. Examination revealed the larynx to be entirely filled with papillomata. These were removed with forceps under general anesthesia on February 24, 1950 and he was placed on 300 mgm. aureomycin daily for 21 days. On April 10, 1950 the larynx was definitely improved with only one small area of papilloma still present; this was removed with forceps and a second course of aureomycin instituted. On May 19, 1950 examination revealed a small amount of papilloma on the right cord but the voice was comparatively clear.

J. H. (Case 107), a one year old white female, was first seen in January 1950 with a history of hoarseness and difficulty breathing since two months of age. Direct examination of the larynx revealed the airway to be markedly limited by papillomatous material along both cords and in the anterior commissure. Under general anes-

TABLE IV.—DOSAGE AND RESPONSE TO AUREOMYCIN THERAPY.

Name	1st Course Dose per kilo. per day	1st Course Duration	2nd Course Dose per kilo. per day	2nd Course Duration	Result
L. K. Case No. 92	50 mgm.	18 days	....	....	Good
H. T. Case No. 104	50 mgm.	16 days	....	....	Good
J. S. Case No. 61	50 mgm.	14 days	50 mgm.	21 days	Poor
L. B. Case No. 106	25 mgm.	28 days	....	....	Good
R. DuR. Case No. 109	25 mgm.	21 days	25 mgm.	21 days	Good
J. H. Case No. 107	25 mgm.	14 days	50 mgm.	21 days	Good
M. L. Case No. 105	50 mgm.	14 days	....	....	Poor

thesia the papilloma were removed and 200 mgm. of aureomycin daily was begun and administered for two weeks. Re-examination revealed considerable papilloma to still be present, and a second course of 200 mgm. daily was advised. During this second course the child vomited after each dose and the mother stopped medication after six days. After a rest for three weeks the larynx was cleared by forceps removal and the child was given 500 mgm. of aureomycin per day for 21 days. On May 12, 1950, one week after the last medication, the larynx was re-examined and only one small area of papilloma was seen which was removed.

M. L. (Case 105), 1½ year old white male infant, had been tracheotomized and was under treatment elsewhere for laryngeal papilloma. Numerous forceps removals had been done because of frequent recurrences of papilloma. He received 50 mgm. of aureomycin per kilo. per day for two weeks and definite improvement in the condition of the larynx was noted. Subsequent examination six weeks later showed the larynx to be free of papilloma although the airway was obstructed by a fusion of the anterior portion of the cords. This result seemed encouraging because the infant had not been free of papilloma since the onset of the disease. However, papilloma were found to have recurred on subsequent examinations which were made in an effort to dilate the stenosis. Three months later (five months after the two weeks' course of aureomycin) re-

examination showed a return of the exuberant papilloma of the larynx. A pedunculated papilloma was found on the lateral pharyngeal wall at this time, and further papillomatous growths were found in the trachea and right bronchus. It is felt that the aureomycin therapy in this child has been inadequate and a repetition of the therapy over a longer period of time is indicated.

The foregoing case histories suggest that aureomycin therapy may be of benefit in the treatment of laryngeal papilloma. It is obvious that the length of time since the institution of treatment is far too brief to draw final conclusions. Dosage administered consisted of 25-50 milligrams per kilogram per day in divided doses, the medication given for a period of 14 to 21 days. The course may be repeated if necessary. The optimum dosage, duration of treatment and interval between courses of aureomycin administration have not yet been apparent from observation of the effects of this agent on this small group of seven cases. However, it would appear that dosage of 50 mgm. per kilo. per day is indicated, and that therapy should be carried out for at least 21 days. It is used only as an adjunct to forceps removal. In those cases in which a good result was obtained, the mucosal surfaces of the larynx appeared smoother than in patients in whom forceps removal has been used alone.

SUMMARY

1. A series of 109 cases of papilloma of the larynx is presented. There are 59 males, 50 females. In 54 patients symptoms developed during childhood (below 16 years of age) and in 55 during adult life. One hundred and two of the patients were white, seven colored. The papilloma were single in 45 of the patients (in ten children and 35 adults) and multiple in 64 (44 children and 20 adults).

2. Etiologic factors of papilloma considered are a filterable virus, chronic irritation and a hormonal agent. The transferability of the growth by means of a filterable agent and the response to aureomycin herein described support the first possibility. Similar lesions elsewhere in the body, notably plantar warts caused by local irritation, support the second possibility. The frequent spontaneous disappearance of the lesions at puberty, the absence of papilloma during each of three pregnancies and recurrence with the resumption of menstruation in one of our patients, and the reported response to estrogenic administration support the third etiologic possibility.

3. The pathology of papilloma of the larynx is discussed. Only true papillomas having the typical stalks of a vascular connective tissue core covered by stratified squamous epithelium in many layers with secondary and even tertiary stalks are included in this series. No histologic difference between the papilloma of children and those of adults was noted although a clinical difference of distribution and texture appears to be present. In spite of frequent recurrence of the papilloma in both the children and the adults and the histologic evidence of growth activity shown by the many cells in mitosis, no patient with papilloma of the larynx in this series developed a carcinoma at the site of the papilloma.

4. The symptoms of papilloma of the larynx are voice change, hoarseness increasing to aphonia and the respiratory changes of gradually increasing respiratory obstruction with cough, stridor, dyspnea, cyanosis and asphyxia. Personality changes and compensatory phenomena such as an elevated red blood cell count are seen in long-standing untreated cases. The diagnosis is made from the mirror or direct examination of the larynx, confirmed by biopsy.

5. Various forms of therapy used are discussed. The most frequent used in this series was forceps removal through the direct laryngoscope. Nineteen of the 109 patients required tracheotomy because of laryngeal obstruction. Sixteen of the 19 patients tracheotomized were under 16 years of age. Of the remaining three, one required tracheotomy because of laryngeal stenosis following x-ray therapy elsewhere and two because of extensive cautery of laryngeal papilloma elsewhere. The local application of podophyllin in two patients appeared to cause a proliferation of the growths. X-ray therapy was used in one adult because of frequent recurrence that was not satisfactorily controlled by simple forceps removal.

6. A preliminary report is made of seven children in this series who have received aureomycin following forceps removal of papilloma. This agent was used because of its specificity for virus disease. The results are encouraging but the length of time since the onset of treatment is too brief to draw final conclusions.

REFERENCES

1. Crowe, S. J., and Breitstein, M. L.: Papilloma of the Larynx in Children, Archives of Surgery 4:275-299 (Mar.) 1922.

2. New, G. B., and Erich, J. B.: Benign Tumors of the Larynx; Study of 722 Cases, Archives of Otolaryngology, 28:841-910 (Dec.) 1938.

3. Ferguson, C. F., and Scott, H. W., Jr.: Papillomatosis of the Larynx in Childhood; Report of 15 Cases, New England Journal of Medicine, 230:477-482 (April 20) 1944.

4. Ullman, E. V.: On the Aetiology of the Laryngeal Papilloma, Acta Otolaryngologica, 5:317-338, 1923.

5. Shope, R. E., and Hurst, E. W.: Infectious Papillomatosis of Rabbits with Note on Histopathology, Journal of Experimental Medicine, 58:607-624 (Nov.) 1933.

6. Lewis, R. M.: A Study of the Effects of Theelin on Gonorrheal Vaginitis in Children, American Journal of Obst. & Gynecology 26:593-599 (Oct.) 1933.

7. Reichert, J. L., Epstein, I. M., Jung, R., and Colwell, C. A.: Infection in the Lower Part of the Genital Tract in Girls, American Journal of Diseases of Children 54:459-495 (Sept.) 1937.

8. Broyles, E. N.: Treatment of Laryngeal Papilloma in Children, Southern Medical Journal 34:239-242 (Mar.) 1941.

9. Jackson, C., and Jackson, C. L.: The Larynx and its Diseases, Philadelphia, W. B. Saunders Co., 1937, p. 377.

10. Cunning, D. S.: Diagnosis and Treatment of Laryngeal Tumors, Journal of the American Medical Association 142:73-76 (Jan. 14) 1950.

11. deSanson, D. R., and Amarante, R. C. L.: Pre-cancerous Formations of the Larynx, Revista Brasileira de Medicina, 5:572-577, 1948.

12. Foster, J. H.: Papillomatosis Laryngis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 42:548-559 (June) 1933.

13. Cohen, L.: Treatment of Intractable Laryngeal Papilloma in Adults with Case Report, Southern Medical Journal 26:621-625 (July) 1933.

14. Broyles, E. N.: Personal communication.

15. Barretto, P.: Personal communication.

16. Koch, J.: Radiation in Laryngeal Papilloma, Archiv fur Ohren-Nasen-und Kehlkopfheit Kunde Sowie die Angrenzenden Gebeite 142:83-96, 1936.

17. Pfeiffer, W.: Therapy of Laryngeal Papilloma, Zeitschrift fur Laryngologie, Rhinologie und Otologie und ihre Grenzgebiete, 23:280-299, 1932.

18. Rosti, E.: Roentgen and Radium Therapy of Laryngeal Papilloma, La Radiologia Medica, Revista Mensile, 25:547-554 (June) 1938.

19. Salinger, S.: Arrested Development of the Larynx Following Irradiation for Recurring Papillomas, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 51: 273-277 (Mar.) 1942.

20. Clerf, L. H.: Laryngeal Complications of Irradiation, Archives Otolaryngology 6:338-345 (Oct.) 1927.

21. Evans, J. D.: Personal communication.

LII

MUCUS CYST OF THE ESOPHAGUS

REPORT OF A CASE AND REVIEW OF THE LITERATURE

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AND

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Benign tumors of the esophagus are of special interest because of their rarity. Of these, mucus cysts appear less frequently than most. Because of this fact this case is presented and added to the literature. Benign tumors may attain considerable size without the production of esophageal symptoms and they are extremely amenable to treatment. Malignant tumors occur more frequently and are quite resistant to treatment.[1]

Their rarity is well illustrated by a study undertaken by Patterson[2] who reviewed the literature from 1717 to 1932 and found only 62 cases of benign esophageal tumors reported, including those cases in which the tumor was first recognized at autopsy. Vinson, Moore and Bowing,[3] in a review of 4000 cases of dysphagia seen at the Mayo Clinic, reported only three cases due to benign tumor of the esophagus. Since their report approximately 7000 additional cases of dysphagia seen at the Mayo Clinic have been reviewed and in this group 12 additional cases of benign tumors of the esophagus were found, bringing the total to 15 out of 11,000 patients seen for dysphagia.[1] Of these 15 cases one case was a mucus cyst of the extramucosal type.

Moersch and Harrington[1] stated, " . . . it is well known that many benign tumors of the esophagus do not give rise to symptoms. This is well exemplified in a review of 7,459 postmortem examinations performed at the Mayo Clinic, in which 44 benign tumors of the esophagus were found. In none of the cases was there a history of esophageal difficulty." In this series the types of tumors found

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were leiomyomas 32, papillomas 3, hemangiomas 3, polypi 2, cysts 2, mucocele 1, and neurofibroma 1.

According to Bockus,[4] "The most common varieties of benign tumors of the esophagus are polypi, lipomas, myomas and fibromas. The benign tumors less frequently found are papillomas, angiomas, hematomas, lymphangiomas and various cysts. The cysts occurring in the esophagus may be parasitic, dermoid, congenital or retention cysts of the esophageal glands. At or near the bifurcation of the trachea is a rather common location for cysts attributed by Patterson to be the result of esophageal diverticuli."

Phelps[5] states that cysts of the esophagus occur as congenital anomalies but are exceedingly rare. He quotes a case described by Buttenweiser, a cyst of the anterior and right wall of the esophagus the size of a pigeon's egg that was found at autopsy on a seven-day-old infant.

For purposes of description Moersch and Harrington[1] divide benign tumors of the esophagus into two types depending upon their site of origin. Those arising from the mucosa or submucosa are commonly known as intra-esophageal or mucosal tumors. Those having their origin in the outer coats of the esophagus are described as intramural, extramucosal lesions.

A cyst of the esophagus of glandular origin found at autopsy is described by Muner;[6] Cuchieri[7] describes the glandular origin of large cysts of the esophagus.

REPORT OF A CASE

A 64-year-old white male was first seen in the out-patient department of the Boston City Hospital, with a chief complaint of dysphagia of three months' duration. He stated that at first solids and then liquids seemed to stick in his throat, and he lost 14 lb. in weight during that period. He was admitted to the hospital for further observation and examination.

Physical examination was essentially negative except for pulmonary emphysema seen by x-ray. Fluoroscopic examination of the esophagus after a barium swallow was reported negative by the x-ray department.

An esophagoscopy was performed using a 30-cm Roberts type esophagoscope under topical 2% pontocaine anesthesia. A mass about 1½ in. in diameter was seen arising from the posterior wall of the esophagus, just below the entrance. It appeared red, congested and rather soft in consistency to palpation with the tip of the esopha-

goscope. The exact identity and origin of this mass could not be determined and the esophagoscope was removed. A second examination under topical 2% pontocaine anesthesia was made five days later and the same mass was found to be present but not as red. At this examination we thought that there seemed to be fluid within the mass but what it was we did not know. Still afraid of puncturing into the mediastinum through the posterior wall of the esophagus, we inserted a hypodermic needle grasped with the edge of a long pair of forceps but no fluid escaped from the mass. The patient was returned to the ward. By this time we suspected a cyst and a third examination was done under topical anesthesia. This time we took a biopsy forceps and bit off a piece of the anterior wall of the mass. There was an immediate escape of about half a teaspoonful of yellow, gelatinous fluid followed by a collapse and disappearance of the mass. Pathological examination of the tissue was reported as epithelial inclusion cyst.

The patient was discharged from the hospital four days later on a soft solids diet. The dysphagia had completely disappeared. He was followed in the out-patient department and his weight had returned to normal with complete absence of the dysphagia. Three months later he complained of postprandial distress. Another esophagoscopy was performed and there was no evidence of the lesion recurring. The esophageal lumen was normal. He was then turned over to the medical service for treatment and was seen six months later when he showed no dysphagia or postprandial distress. All symptoms had disappeared under the medical regime.

SUMMARY

1. Benign tumors of the esophagus are comparatively rare; mucus cysts are among the rarest of the benign tumors.

2. Benign tumors of the esophagus may attain considerable size without producing symptoms.

3. They are extremely amenable to treatment.

4. Sixty-two cases of benign tumors of the esophagus were reported in the literature from 1777 to 1932.

5. In a review of 11,000 consecutive cases of dysphagia at the Mayo Clinic, 15 were found to be due to benign tumors of the esophagus.

6. The types of benign tumors found are leiomyoma, papilloma, hemangioma, polyp, cyst, mucocele, and neurofibroma.

7. Anatomical types are intra-esophageal or mucosal, and extramucosal or intramural.

8. A case of dysphagia due to an epithelial inclusion cyst is reported.

18 Broad St.

REFERENCES

1. Moersch, H. J., and Harrington, S. W.: Benign Tumors of the Esophagus, Annals of Otology, Rhinology and Laryngology 53:800-817 (Dec.) 1944.

2. Patterson, E. J.: Benign Neoplasms of the Esophagus; Report of a Case of Myxofibroma, Annals of Otology, Rhinology and Laryngology 41:942-950 (Sept.) 1932.

3. Vinson, P. P., Moore, A. B., and Bowing, H. H.: Hemangioma of the Oesophagus, Am. J. M. Sc. 172:416-418 (Sept.) 1926.

4. Bockus, H. L.: Gastro-Enterology, Philadelphia, W. B. Saunders Co., 1946.

5. Phelps, Kenneth A.: Congenital Anomalies of the Esophagus with a Report of Nine Cases, Annals of Otology, Rhinology and Laryngology 39: 364 (June) 1930.

6. Muner, I.: Cysts of Glandular Origin; Autopsy of a Case, Gioa. Venito di Sc. Med. 10:613-623 (Sept.) 1936.

7. Cuchieri, E.: Glandular Origin of Large Cysts of the Mucus Membrane, Pathologica 23:617-623 (Oct. 15) 1931.

LIII

RHINOSCLEROMA

REPORT OF A CASE TREATED WITH DIHYDROSTREPTOMYCIN

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AND

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Although rhinoscleroma is uncommon in this country, isolated cases may be encountered anywhere, and it behooves the rhinologist to recognize it or at least to suspect it.

Our patient is a white male, 57 years old. He was born in Kiev, Russia, and emigrated to this country in 1912. The subsequent medical history is sketchy for the patient cannot well remember dates or time sequences.

Between 1915 and 1920 he was treated at a clinic for nasal obstruction and crusting about the alae. It is interesting to speculate whether or not the disease was in its incipient stages 38 years ago; however, no description is obtainable.

In the early 1920's the patient remembers that he had to abstain from drinking citrus juices and alcohol because of the intense burning that would ensue. On November 5, 1938, he was seen by one of us (S.M.M.) and the following findings were noted: A nodular mass was present anteriorly on the floor of the right nostril, the left inferior turbinate was replaced by a white fibrous mass, a thin secretion was present in both nostrils, the mucosa of the palate was covered with a thin grayish membrane, bleeding followed the least attempt to remove the membrane. A study was begun but the patient did not return. It is interesting to note that he had been referred by a dentist who wished to extract the remaining teeth.

From the Norwegian American Hospital and the Department of Rhinology, Otology and Laryngology, University of Illinois College of Medicine, Chicago, Illinois.

Read at the meeting of the Chicago Laryngological and Otological Society, Chicago, Illinois, February 6, 1950.

In 1940 the patient was seen by an internist who thought the membrane suggestive of Vincent's stomatitis. A smear was reported, by the state laboratory, as positive for Vincent's organisms. Fowler's solution and neo-salversan were tried without success.

In May, 1945, the patient was hospitalized by another rhinologist and the obstructing masses at the nares cut away. The pathologist's report was chronic inflammatory tissue with a strong possibility of malignancy.

Two years later the patient was again hospitalized by the same rhinologist for relief of the obstruction. The ear, nose and throat findings (by the intern) were "nasal mucosa markedly elevated, serous crusting present;" no mention was made of the mouth. The pathologist's report this time was chronic inflammatory tissue with the presence of foam cells and Unna bodies. A diagnosis of rhinoscleroma was suggested.

Sometime between 1945 and 1947 x-ray therapy was given. The patient could not remember the name of the roentgenologist and so the type and quantity of therapy can not be determined.

On July 2, 1949 he returned to our office. The following physical findings were noted: Both nares were almost entirely occluded by firm, reddish nodules which so constricted the anterior nares that a nasopharyngoscope could barely be passed. These fibrotic changes extended back about one inch, the posterior portion of the nose and the whole of the epipharynx was clear. The upper lip was thickened and stiffened by subepithelial infiltration. The gingival tissue of the upper alveolar ridge was hyperemic and hyperplastic. All but the upper incisors had been extracted, the whole of the hard and soft palate, including the tonsillar pillars was covered by a dirty yellowish exudate with a foul smell. Upon wiping this away a series of shallow, hyperemic ulcers were exposed, some were discrete, others confluent; the hypopharynx, larynx and trachea were negative.

A presumptive diagnosis of rhinoscleroma was made. The previously mentioned biopsies were traced down and the diagnosis of rhinoscleroma was confirmed. Then following the report of Levine and Hoyt[1] bacteriological studies were undertaken. A gram-negative capsulated bacillus was isolated which fulfilled the gross and cultural characteristics ascribed to *Klebsiella rhinoscleromatis*. Further tests were made to determine the sensitivity of this bacillus to dihydrostreptomycin. A dilution of 0.39 micrograms (4/10,000,000) completely inhibited growth. An audiogram was made and a caloric test, using 5.0 cc of ice water was performed. The patient was

hospitalized and dihydrostreptomycin was given in the following amounts:

7-14-49 to 7-18-49	0.25 gm	q.i.d.
7-19-49 to 7-27-49	0.75 gm	b.i.d.
7-28-49 to 8- 6-49	0.5 gm	s.i.d.
8- 7-49 to 8-17-49	0.75 gm	s.i.d.

The following progress notes indicate the rapidity of recession of the scleromatous lesions:

July 16. No change in palate, perhaps slight recession of granuloma at alveolar margin.

July 17. Patient states he feels better; findings the same as before with possible exception of lessening of hyperemia.

July 19. Subjective improvement is much greater than clinical evidence warrants.

July 20. Inflammatory reaction subsiding.

July 22. Nasal lesion subsiding; palate looks better.

Thus eight days after beginning streptomycin therapy there was a great subjective improvement and a definitely discernible change in the physical findings. On August 1, bacteriologic studies showed no growth on the usual media. Therapy was continued on an out-patient basis from July 27 to August 17. Caloric tests performed during this time and subsequently showed no vestibular involvement. The audiogram remained essentially the same.

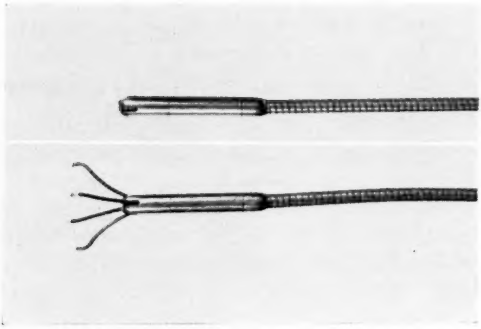
To date all of the lesions have either subsided or disappeared with the exception of one ulcer on the hard palate. The patient is going to have his upper incisors extracted and it is hoped that the protection of the prosthesis may help the ulcer to heal.

55 East Washington Street.

REFERENCE

1. Levine, M. G., and Hoyt, R. E.: Diagnosis of Scleroma, Arch. Otolaryng. 47:438 (Apr.) 1947.

New Instrument



LIV

INSTRUMENT FOR REMOVING FLATHEAD SCREWS, NAILS OR TACKS

A. C. Stutsman, M.D.

St. Louis, Mo.

The adaptation of a forcep developed for the removal of calculi from the ureter has been successfully used to remove a flat-headed screw from the tracheobronchial tree. It could be used for beads and other globular objects as well as for tacks and nails when the head is presenting.

The instrument is small enough to go through a 4-mm bronchoscope. The prongs are made of cobenium and are sturdy enough to exert pressure against the edematous wall when opened. The four prongs are extended as the forcep is opened in a position to engage the foreign body without further introduction of the instrument. The flexible shaft gives mobility to the tip although the shaft may be had semirigid or rigid.

The forcep is made by The Phillips-Drucker Company, 2245 South Vandeventer Ave., St. Louis, Missouri.

640 Beaumont Building.

From the Department of Otolaryngology, Washington University School of Medicine, St. Louis, Missouri.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, February 6, 1950

THE PRESIDENT, DR. OLIVER E. VAN ALYEA, IN THE CHAIR

Tumors of the Trachea

PAUL H. HOLINGER, M.D.

FRANK J. NOVAK, III, M.D. (By Invitation)

AND

KENNETH C. JOHNSTON, M.D. (By Invitation)

CHICAGO, ILL.

(Abstract)

Tumors of the trachea are relatively rare. According to various authorities they range in frequency from 1-300 to 1-800, as compared to laryngeal tumors. They are usually not recognized until they are far advanced because no localizing symptoms develop until they become large enough to produce severe respiratory obstruction.

Thirty-seven cases of tracheal tumors seen at the Research and Educational Hospitals, St. Luke's Hospital and Children's Memorial Hospital during the past 14 years are reviewed. They may be classified as nonspecific and specific inflammatory tumors, and benign and malignant neoplasms. Twenty-four of the 37 tumors were primary in the trachea. The remaining 13 tumors invaded the trachea from adjacent structures to give signs, symptoms and endoscopic findings of primary tracheal tumors. Five of the tumors were inflammatory; three were nonspecific post-tracheotomy granulomas which caused respiratory obstruction in infants and children and prevented extubation or required endoscopic removal after extubation; the granulomas developed at the point of the tracheostomy. One specific inflammatory tumor was a large tuberculoma that, together with tuberculous ulceration and exudate eventually caused the death of the patient from tracheal obstruction. A second specific inflammatory tumor due to torula which obstructed the trachea and right bronchus of a six-year-old boy. It was removed endoscopically.

Of the neoplastic tumors, six were benign and 26 malignant. Of the six benign tumors two were amyloid tumors and one each a

papilloma, fibroma, xanthoma, and adenoma. Ten of the malignant tumors were squamous cell carcinomas, one a cylindroma, two myosarcomas, and 13 were carcinomas of adjacent structures invading the trachea to give primary symptoms and endoscopic findings of tracheal tumors. Five of these were thyroid carcinomas, five bronchogenic carcinomas on or close to the carina to involve both bronchi, and three were esophageal carcinomas with extensive tracheal invasion which caused respiratory obstruction.

In most cases in this series the first symptom noted was a hacking, dry, constantly irritating cough that could not be relieved with cough sedatives or expectorants. The cough was frequently associated with hemoptysis which varied from slight blood streaking to gross hemorrhage, most pronounced in the malignancies. Stridor was another constant symptom which at first was quiet and positional and later became a loud, asthmatic expiratory wheezing which was bilateral, and so clearly simulated asthma that most of the patients had had thorough and repeated allergy studies and treatment for asthma. Dyspnea and orthopnea were present in the late cases, and hoarseness occurred in the malignancies when vocal cord paralysis developed because of outward extension or mediastinal involvement. Dysphagia was occasionally noted.

The history of symptoms noted above and the physical findings were exceedingly important in establishing the diagnosis. The x-ray studies included inspiration and expiration chest films, lateral x-ray films of the neck and trachea, planograms in the anteroposterior and lateral planes, and bronchographic studies. The final diagnosis was made by bronchoscopic examination and biopsy.

The first problem in treatment was usually establishment of the airway. This frequently was an emergency procedure consisting of coring the tumor, removal of obstructive tissue by forceps or snare, and occasionally a tracheotomy, the tube being inserted below the tumor or beyond it by the use of a long tracheotomy tube. In subsequent endoscopic removal electroresection and coagulation were important endoscopic procedures in keeping the airway free of tissue. Radon implantation and external irradiation were adjuncts in the treatment of the malignancies. Tracheal resection was accomplished in one case of myosarcoma of the trachea to give a five-year cure.

DISCUSSION

DR. FRANK J. NOVAK, III: I would like to show the histologic picture of four tumors not in the series of slides just shown. The first is an amyloid tumor, which ordinarily is difficult to demonstrate because it has such an amorphous structure. Congo red was used to supravitally stain the amyloid.

The next is a squamous cell tumor. This patient is living approximately six and a half years from the time of the original diagnosis.

The last slide shows papilloma; this patient also has chronic lymphatic leukemia, with 33,000 white cell count at this time.

DR. A. M. LAZAR: To Dr. Holinger's excellent presentation I should like to add the case of a tumor of the trachea in an infant 20 months of age, finally established as a hemangioma. In this instance we had the co-operation of the pediatrician, which is not always possible. The child came in with an improper diagnosis, but too often the laryngologist is not called until it is a serious emergency. She had been treated in the dispensary for laryngotracheobronchitis for three weeks, and was finally admitted to the hospital when she had extreme difficulty in breathing. She was under the care of the Pediatric Service for 36 hours, the symptoms did not improve, and the nose and throat department was called. Not having seen the child previously or having the opportunity of making an examination, the diagnosis of tracheobronchitis was accepted. A tracheotomy was performed and the child got immediate relief; she was given the usual therapy for laryngotracheobronchitis. Decannulation on the third day was tried without any benefit and had to be repeated several times. A laryngoscopy was performed but was not revealing. On the following day we did a bronchoscopy and found what looked like a granulomatous mass on the tracheal infraglottic area. The child did well for several days with the tube in place, then one of our senior attending men had the idea that she might be "tube fast." The tube was then removed and the child died about 12 hours later of asphyxia.

At autopsy a congenital hemangioma below the left vocal cord was found. This demonstrates that diagnosis should be made early. This is the ninth case recorded in the last 27 years, and of the eight patients reported previously, seven died of asphyxia or following surgery. This case of hemangioma of the larynx teaches us also that to leave a tube in a child's trachea for an indefinite period will definitely do no harm to the child.

Rhinoscleroma: Case Report

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AND
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CHICAGO, ILL.

(Summary)

The salient facts about scleroma and rhinoscleroma are briefly reviewed.

Scleroma and rhinoscleroma have always been endemic in central and southeastern Europe, and now also in South and Central America.

Sporadic cases of this disease may be encountered anywhere in the United States and Canada.

The rhinologist particularly should be alert to the possible existence of an isolated case of rhinoscleroma in this country and be familiar with its diagnostic features.

The disease of scleroma in all its forms is considered to be mildly contagious.

Until recently x-ray and radium gave the best therapeutic results, but were not completely satisfactory.

A modification of streptomycin, called dihydrostreptomycin, now available, is found to be relatively nontoxic to the vestibular portion of the labyrinth and is as effective therapeutically as streptomycin.

A case of rhinoscleroma is reported, with definite rapid clinical and subjective relief following the use of dihydrostreptomycin.

DISCUSSION

DR. S. M. MORWITZ: A chronic disease such as rhinoscleroma with common manifestations and uncommon occurrence is a subject which should be called to our attention from time to time. I will frankly admit that when I first saw this patient 12 years ago the thought of this disease never entered my head. We usually consider in differential diagnosis, acid-fast lues or possible malignancy. I think the effect of dihydrostreptomycin in this condition probably offers more hope than anything else, if the disease can be diagnosed in the early stage, in which case the drug might effect a real cure instead of merely improvement.

DR. JACK WEISS: This has been an excellent exposition of the salient features of rhinoscleroma. If I may be permitted a personal historical note, I should mention that this topic was the subject of my candidate's thesis before this Society in December, 1938. ("Scleroma (Rhinoscleroma): Histologic Changes Following Teleradium Therapy. Review of Scleroma in the United States.")

This paper reported two cases, plus 58 cases collected from the American literature. In recent years a substantial additional number of cases have been added. I believe the increase to be due in large part to increased clinical alertness and diagnostic acumen.

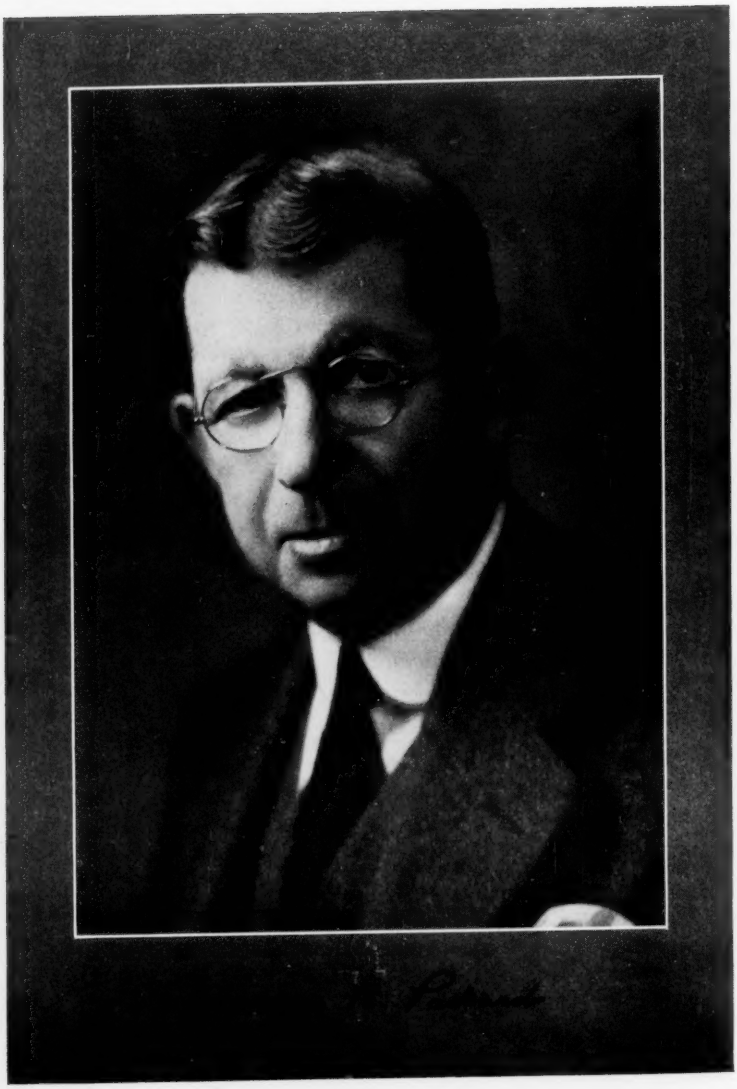
One of the cases treated in 1936 was a 55-year-old Mexican woman who had bilateral nasal lesions which began in 1915. She received 32,000 milligram hours. The lesions regressed greatly. Patency of the nares was restored surgically. The lesions remained inactive for about 11 years. Evidence of recurrent activity was then observed. However, she refused dihydrostreptomycin therapy, as the airway remains adequate.

The granddaughter of this patient may be cited as an additional instance of indigenous scleroma. This case will be reported in full at a later date. When first examined in 1942, she was found to have bilateral, partially stenotic nasal lesions. Microscopic examination revealed the typical structure of scleroma; nasal culture yielded Frisch's bacillus. She received two courses of autogenous vaccine with no notable result. During my absence in military service, subsequent management included electrosurgical opening of the nares and three courses of irradiation. Partial regression was followed by recurrence. When seen in 1947 she exhibited almost total bilateral nasal stenosis, plus laryngeal and subglottic lesions. Irradiation to the nose and larynx gave little improvement. Respiratory obstruction necessitated tracheotomy. Between June and October, 1947, she received three courses of streptomycin, totalling 112 gm. The response was gratifying. The laryngeal lesions regressed completely on gross examination. The nasal lesions shrunk and a subsequent plastic correction and skin graft restored the patency of the nares. Transient labyrinthine disturbance occurred during therapy. A recent culture of the larynx failed to show *Klebsiella rhinoscleromatis*.

Scleroma is being seen with increasing frequency in this country. The nasal lesions should be easily recognized and confirmed by laboratory aids. Laryngeal lesions may be more confusing and differentiation must be made from other diseases, especially syphilis.

DR. FRANCIS LEDERER: I think it might be a good thing to go to Mexico, because here we see these cases so seldom that they defy diagnosis when they do appear. In Mexico there is one series of 400 cases and another of 300 reported.

The only point one would have in presenting a discussion of rhinoscleroma in Chicago is that in Mexico they are worried about the fact that their farm population is drifting to the United States. They say they are losing 100,000 good men a year. It is quite possible, then, that we may see more of these lesions and may consider them chronic nonspecific granulomata. If this presentation is of any value it will make us think of rhinoscleroma in a differential diagnosis.



FRANCIS RANDOLPH PACKARD, M.D.

1870-1950

Francis R. Packard was born March 23, 1870 at Philadelphia, Pennsylvania, and died there on April 18, 1950.

He was the son of John Hooker Packard, Surgeon to the Pennsylvania and the Episcopal Hospitals, and Elizabeth Wood Packard. He attended Rugby Academy in Philadelphia, and graduated from the Biological Department of the University of Pennsylvania in 1889 and from the Department of Medicine in 1892. After serving as Resident Physician in the Childrens Hospital and later in the Pennsylvania Hospital he began the private practice of medicine in 1895, three years later confining his work to the ear, nose and throat.

For some years he was Professor of Laryngology and Otology at the Philadelphia polyclinic and the College for Graduates in Medicine, and served on the Staffs of several other local hospitals and eleemosynary institutions.

In 1899 he was married to Christine Curwen, and some years after her death, in 1901 to Margaret Horstman. Three daughters were born of this marriage.

Doctor Packard was a member of many social, medical, literary and historical societies. He held many posts of honor in the College of Physicians of Philadelphia, and became its President in 1930. In this year he was likewise President of the American Laryngological Association. He was a Fellow of the American Otological Society and the American Laryngological, Rhinological and Otological Society. For a short time he served as First Lieutenant and Assistant Surgeon in the Spanish-American war, and during the First World War was attached to British General Hospital No. 16 later serving as Chief Centre Consultant in Laryngology and Otology for the District of Paris.

Between 1897 and 1916 Dr. Packard wrote a score of articles on otolaryngological subjects, but he is best known for his contributions to the literature of medical history and to the history of medical literature. He was at one time Editor of the American Journal of the Medical Sciences, and after 1917 Editor of the Annals of Medical History.

In his seventy-second year, and eight years before his death he received the Newcomb Award of the American Laryngological Association for distinguished contributions to the literature of the history of medicine.

Abstracts of Current Articles

NOSE

Contribution to the Question of Granuloma Gangraenescens.

Bergquist, B., and Kock, H.: Acta Oto-laryng. 37:415 (Oct.) 1949.

The authors report two cases of granuloma gangraenescens or malignant granuloma involving the nose and upper jaw. From their observations they conclude that the process is primarily neoplastic, possibly complicated by fusospirochetal symbiosis. Histological studies in one case suggested reticulum cell sarcoma and roentgen therapy seemed to result in regression of the process.

While the authors stress susceptibility to irradiation, this may be open to doubt, as the outcome in both cases was fatal, as would be expected. The evidence presented fails to justify the assumption of malignancy rather than the more generally accepted theory of anaerobic infection of undetermined origin.

HILL.

LARYNX

Hemangiomi Laryngis.

Berntsen, W.: Acta Oto-laryng. 37:420 (Oct.) 1949.

Two cases of hemangioma of the larynx are reported. One patient complained only of hoarseness. No treatment was given and the laryngeal picture was unchanged for six years. The second patient had a large hemangioma causing increasing dyspnea. Laryngofissure was performed but the tumor was considered inoperable and tracheotomy was done to relieve obstruction. Five days later death ensued from a spontaneous hemorrhage.

One cannot help wondering why some effort to destroy the tumor, possibly by electrocoagulation or by application of radium or radon, was not employed after the interior of the larynx had been exposed by thyrotomy.

HILL.

ESOPHAGUS

Spontaneous Perforation of the Esophagus.

Lynch, J. J.:New England J. Med. 241:395 (Sept. 15) 1949.

Three cases of spontaneous perforation of the esophagus are reported, apparently due to violent vomiting. When this is followed by severe pain in the lower chest or upper abdomen spontaneous perforation should be suspected. Cervical subcutaneous emphysema is diagnostic, as is the roentgen finding of air in the mediastinum. The author advocates early operation to close the perforation and relieve compression of the mediastinum, together with pleural drainage and antibiotic therapy.

HILL.

BRONCHI

Delayed Pneumonia and Urticaria Following Bronchography.

Barr, H. E.: New England J. Med. 240:505 (Mar. 31) 1949.

A number of fatal cases following bronchography have been reported, in which postmortem examination revealed both lungs atelectatic and the tracheobronchial tree completely filled with tenacious mucus. These have been considered to be allergic in origin and attributed to the iodine in the contrast media. The use of lipiodol in allergic individuals has been followed by delayed pneumonia, together with urticaria and blood eosinophilia. This delayed reaction resembles the delayed reaction of serum sickness. The author advises that iodine-sensitivity tests be performed on all patients with any history of allergy before bronchography is considered; and that this be performed only if the information to be gained is essential, where the test is positive. Overfilling should be avoided and the bronchi should be emptied as much as possible by postural drainage after the examination is completed. Cases should be under observation for at least two weeks. Antihistaminic drugs may be beneficial. One case is reported in which pneumonia developed 14 days after instillation of lipiodol.

HILL.

MISCELLANEOUS

Acute Herpetic Gingivostomatitis in the Adult.

Rogers, A. M., Correli, L. L., Black, H., and Scott, T. F. M.: New England J. Med. 241:330 (Sept. 1) 1949.

The clinical picture is similar to that seen in infants and characterized by an acute febrile course with multiple aphthous ulcerations of the mouth and pharynx, marginal gingivitis and cervical adenopathy. The diagnosis may be confirmed by isolation of the *herpes simplex* virus from the saliva, the finding of typical inclusion bodies in a biopsy specimen and the appearance of specific neutralizing antibodies during convalescence. Three cases are reported.

HILL.

Books Received

Surgical and Maxillofacial Prosthesis.

By Oscar Edward Beder. Pp. viii+51, with 31 figures. Columbia University, New York, King's Crown Press, 1949. (Price $3.00)

Current Therapy 1950. Latest Approved Methods of Treatment for the Practicing Physician.

Editor: Howard F. Conn, M.D. Consulting Editors: M. Edward Davis, Vincent J. Derbes, Garfield G. Duncan, Hugh J. Jewett, William J. Kerr, Perrin H Long, H. Houston Merritt, Paul A. O'Leary, Walter L. Palmer, Hobart A. Reimann, Cyrus C. Sturgis, Robert H. Williams. Pp. xxxii+736. Philadelphia and London, W. B. Saunders Company, 1950. (Price $10.00)

Public School Audiometry: Principles and Methods.

By Loraine Anson Dahl, Formerly Field Supervisor, Wisconsin State-wide Hearing Survery; Supervisor, Hearing Test Service, Speech Clinic, Purdue University; Research Associate, Department of Otolaryngology, University of Iowa. Pp. 290. Danville, Illinois, The Interstate, 1949.

The Nose. An Experimental Study of Reactions within the Nose in Human Subjects During Varying Life Experiences.

By Thomas H. Holmes, M.D., Lester N. Hofheimer Research Fellow in Medicine, Helen Goodell, B.S., Research Fellow in Medicine; Stewart Wolf, M.D., Associate Professor of Medicine; and Harold G. Wolff, M.D., Professor of Medicine (Neurology), Cornell University Medical College, New York. Pp. xvi+154 with 35 figures (5 illustrations in color). Springfield, Charles C. Thomas, 1950. (Price $4.50)

Proceedings of the First Clinical ACTH Conference.

Edited by John R. Mote, M.D., Medical Director, Armour Laboratories. 178 *Contributors.* Pp. 624 with 414 illustrations. Philadelphia and Toronto, The Blakiston Company. 1950. (Price $5.50)

Diseases of the Eye, Ear, Nose, and Throat: A Textbook for Nurses.

By Albert P. Seltzer, M.D., Sc.D. (Med.), F.I.C.S., F.A.C.S. Assistant Professor in Otolaryngology, Graduate School of Medicine, University of Pennsylvania; Assistant Otolaryngologist to the Mt. Sinai Hospital, Philadelphia; Associate Chief in Ear, Nose, and Throat, St. Luke's Medical Center; Consulting Otolaryngologist to the Mercy-Douglas Hospital, Philadelphia; Instructor of Nurses at St. Luke's Medical Center.

With the Technical Assistance of Bernard C. Gettes, M.D. Diplomate, American Board of Ophthalmology; Instructor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Instructor of Nurses in Ophthalmology, Wills Hospital; Chief Ophthalmologist, Stetson Hospital. Pp. xvii +347 with 28 figures. New York, McGraw-Hill Book Company, Inc., 1950. (Price $4.00)

Harvey Cushing: Surgeon, Author, Artist.

By Elizabeth H. Thomson; Foreword by John F. Fulton. Pp. xviii+347 with 12 plates. New York, Henry Schuman Inc., 1950. (Price $4.00)

If You Have a Deaf Child. A Collection of Helpful Hints to Mothers of Deaf Children.

By Mrs. Spencer Tracy; Julius Richmond, M.D.; Mrs. James L. Cassidy; Irene M. Josselyn, M.D.; Francis L. Lederer, M.D.; Harold Westlake, Ph.D.; Maurice V. Moriarty; Isaac Jolles; Jean Utley, Ph.D.; and Daniel T. Cloud. Published for the Illinois Annual School for Mothers of Deaf Children by the University of Illinois Press, Urbana, Illinois, 1949.

Névralgies du Glosso-pharyngien.

By Maurice Deparis, Professeur agrégré à la Faculté de Médecine de Paris; Médecin des Hôpitaux de Paris. Pp. 156 with 5 figures. Paris, Masson et Cie., 1949. (Price 360 fr.)

Bronchography.

By Eelco Huizinga, M.D. and G. J. Smelt, M.D., Department of Oto-rhino-laryngology, University of Groningen. Pp. 270 with about 150 illustrations. Assen, Netherlands, Van Gorcum and Comp. Ltd. (G. A. Hak and H. J. Prakke), Publishers, 1949. (Price about $10.00)

Korrosion des Oesophagus und des Ventrikels. Ihre Folgen und ihre Behandlung.

By U. K. Kiviranta, Helsinki, Finland. Acta Oto-laryngologica Supplementum LXXXI, 1949, pp. 128.

Theories of Hearing: A critical study of theories and experiments on sound-conduction and sound-analysis in the ear.

By P. J. Kostelijk, M.D. Research Committee for Sanitary Engineering T. N. O. Section on Sound. Pp. X+180 with 35 figures. Leiden, Universitaire Pers Leiden, 1950.

Laringoceles.

By Roberto Maisonnave. Pp. 223 with 65 figures. Buenos Aires, El Ateneo, 1947.

Occupational Eye Diseases and Injuries.

By Joseph Minton, F.R.C.S. (Eng.), Ophthalmic Surgeon to the Hampstead General Hospital, Queen Elizabeth Hospital for Children, West End Hospital for Nervous Diseases, London Jewish Hospital. Lecturer on Industrial Ophthalmology at the Royal Institute of Public Health and Hygiene and the Royal College of Nursing, London. Hunterian Professor (1947) Royal College of Surgeons, England. Late Major R.A.M.C. Pp. viii+184 with 24 figures. New York, Grune & Stratton, Inc., 1949. (Price $4.50)

Clínicas y Policlínicas Otorrinolaringológicas.

By Francisco H. Rivero. Pp. 311, illustrated. Havana, Cuba, P. Fernandez y Cía., 1950.

Die Schleimhäute des Ohres und der Luftwege. Biologie und Klinik.

By Dr. Max Schwarz. Pp. viii+140, with 57 illustrations. Berlin, Göttingen and Heidelberg, Springer-Verlag, 1949.

Bronchologie. Technique Endoscopique et Pathologie Trachéo-bronchique.

By Andre Soulas and Pierre Mounier-Kuhn. Pp. 654, with 304 figures and 24 colored plates. Paris, Masson et Cie., 1949. (Price 4000 fr.)

Arachnoidites Opto-chiasmatiques et Maladie Neuro-vasculaire.

By J.-N. Taptas, Ancien chef de clinique neuro-chirurgicale à la Faculté de Paris, Professeur agrégé des Facultés de Médecine; and Th. Dimopoulos, Chef de clinique neurologique de l'Hôpital de la Croix-Rouge de'Athènes. Pp. 110. Paris, Masson et Cie., 1949. (Price 280 fr.)

La Trompe d'Eustache.

By J. Terracol, Professeur à la Faculté de Médecine de Montpellier, Correspondant national de l'Académie de Médecine; A. Corone, Ancien interne des Hôpitaux de Montpellier; and Y. Guerrier, Chef des Travaux anatomiques à la Faculté de Médecine de Montpellier. Pp. 218 with 82 figures and 1 colored plate. Paris, Masson et Cie., 1949. (Price 1200 fr.)

Tonsillectomy as Treatment of Acute Peritonsillitis, with Clinical and Statistical Observations.

By V. Seppo Virtanen, Helsinki, Finland. Acta Oto-laryngologica Supplementum LXXX, 1949, pp. 173.

Pyramideneiterungen. Deren Verhütung und Behandlung.

By Prof. Dr. O. Voss, Frankfurt a.M.—Berchtesgaden. Pp. 256 with 71 illustrations. Stuttgart, Georg Thieme Verlag, 1949. Agents for U.S.A.: Grune & Stratton, Inc., New York.

Notices

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct the following examinations:

October 3-6 in Chicago, Illinois, at the Palmer House.

January 8-11 in New York City, at the Hotel Biltmore.

DEAN M. LIERLE, M.D., *Secretary.*

HOME STUDY COURSES

The 1950-1951 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1st and continue for a period of ten months. Registrations must be completed before August 15th. Detailed information and application forms may be secured from Dr. William L. Benedict, Executive Secretary-Treasurer, 100 First Avenue Building, Rochester, Minnesota.

HARVARD MEDICAL SCHOOL

The Harvard Medical School offers a series of four consecutive graduate courses in otology and laryngology from November 6, 1950, to March 31, 1951.

Courses will be given in Histopathology, Audiology, Anatomy of the Head and Neck and Anatomy of the Temporal Bone.

For further information, apply to Assistant Dean, Courses for Graduates, Harvard Medical School, Boston 15, Massachusetts.

UNIVERSITY OF ILLINOIS

The University of Illinois, College of Medicine, Department of Otolaryngology announces its annual postgraduate course in Basic Otolaryngology beginning October 2, 1950. A full time program of instruction is followed for those intending to prepare for special practice. The course terminates on June 15, 1951.

For information write the University of Illinois, College of Medicine, Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Illinois.

INTERNATIONAL COURSE IN AUDIOLOGY

STOCKHOLM, SEPTEMBER, 1950

An international course in audiology will be given in Stockholm from September 11-20 inclusive. Lectures will come under the following general heads: Anatomical, Physiological, Physical and Psychological Background; Hearing Loss; Hearing Tests; Treatment of Hearing Loss; and Remedies. There will be practical courses and demonstrations in Tone and Speech Audiometry, Selection of Hearing Aids, and Fenestration.

Instruction will be given by some 35 outstanding otologists of Europe, North and South America. For information apply to C. A. Tegnér, Secretary, 33, Birger Jarlsgatan Stockholm C. Sweden. Telegrams: Categner, Stockholm.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION THE AMERICAN MEDICAL ASSOCIATION

(List Corrected to April 1, 1950)

Aurex Model F
Aurex Model H

Beltone Mono-Pac
Beltone Harmony Mono-Pac
Beltone Symphonette
Beltone Mono-Pac Model M

Cleartone Model 500
Cleartone Regency Model

Dysonic Model No. 1

Electroear Model C

Gem Hearing Aid Model V-35

Maico Type K
Maico Atomeer
Maico UE Atomeer
Mears Aurophone Model 200
1947-Mears Aurophone Model 98
Micronic Model 101 (Magnetic Receiver)
Micronic Model 303 (See Silver Micronic)
Microtone T-3 Audiomatic
Microtone T-4 Audiomatic
Microtone T-5 Audiomatic
Microtone Classic Model T9
Microtone Classic Model 45

National Cub Model (C)
National Standard Model (T)
National Star Model (S)

Otarion, Model E-1
Otarion, Model E-1S
Otarion, Model E-2
Otarion, Model E-4
Otarion, Models F-1 & F-2

Paravox Models VH and VL
Paravox Model XT
Paravox Model XTS
Paravox Model Y (YM, YC and YC-7)

Radioear Permo-Magnetic Multipower
Radioear Permo-Magnetic Uniphone
Radioear All-magnetic Model 55

Silver Micronic (Crystal Receiver) Model 101
Silver Micronic (Magnetic and Crystal) Models 202M & 202C (See Micronic)
Silvertone Model 103BM
Solo-Pak Model 99
Sonotone Model 600
Sonotone Model 700
Sonotone Model 900
Sonotone Models 910 & 920
Sonotone Model 925
Super-Fonic Hearing Aid

Televox Model E
Telex Model 22
Telex Model 97
Telex Model 99
Telex Model 200
Telex Model 1700
Tonemaster Model Royal
Trimm Vacuum Tube Model 300

Unex Model A
Unex Midget Model 95
Unex Midget Model 110

Vacolite Model J

Western Electric Model 63
Western Electric Model 64
Western Electric Models 65 & 66

Zenith Model 75
Zenith Miniature 75

All of the accepted hearing devices employ vacuum tubes.
Accepted hearing aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS

Aurex (Semi-Portable) Precision Table Hearing Aid
Sonotone Professional Table Set Model 50